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H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Vol. XCI, N. S. Vol. 45 — August, 1937 — No. 2

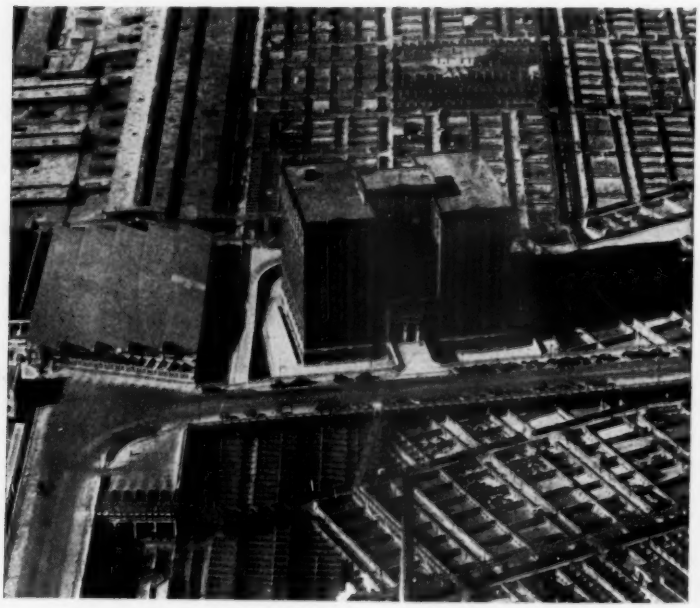
OMAHA IN READINESS

En route, Omaha to Chicago, Aug. 1—Practically the entire day has been spent in conference with the Committee on Local Arrangements, perfecting the details for the program for the 74th annual meeting of the A. V. M. A., which will be held in Omaha during the third week of this month. Veterinarians and their wives, to the number of almost fifty, from points as far east as Ames, Iowa; from Sioux City on the north, and from Lincoln on the west, were in Omaha for the final meetings of their various committees prior to the convention. The chairmen of these groups now report everything in readiness for the two thousand visitors who are expected to attend Omaha's second A. V. M. A. convention. Probably no meeting of veterinarians has had the publicity that the 1937 A. V. M. A. convention has already received and will receive from now on through the week of August 16.

As for the program, suffice it to say that it would be just about impossible to crowd in another thing. It is solid, from the opening session, on Tuesday, until the close of the clinic, Friday afternoon. Monday undoubtedly will see several hundred

members on hand for the meetings of the Executive Board, the House of Representatives, and the various committees. The commercial and educational exhibits promise to be unusually attractive this year. In the form of entertainment we are to have a polo game and a special performance of the Ak-Sar-Ben Den Show, Tuesday evening. The banquet, President's reception and dance will be a three-in-one event for Wednesday evening. A new departure will be the holding of the alumni meetings on Thursday evening. This plan gives more time for getting these groups organized than has been possible under the old plan. The clinic will embrace a number of new features that should prove to be very interesting.

The headquarters hotel—the Fontenelle—is sold out for the week—every room reserved, and several other nearby hotels probably will be by this time next week, judging by the way reservations are being made. Crops in the territory are the best in years, collections have been improving, and veterinarians are feeling better, along with everybody else. It would be hard to find a veterinarian within 500 miles of Omaha who is not planning to be at the meeting.



LIVE STOCK EXCHANGE BUILDING AND UNION STOCK YARDS AT OMAHA. THE OMAHA MARKET RATED THIRD IN THE UNITED STATES, ON THE BASIS OF 1936 RECEIPTS.

Registration

The Omaha convention will be financed along slightly different lines than has been the custom in the past. No veterinarian in the Omaha territory, either individually or collectively, has been asked to contribute to the fund usually raised by the Committee on Local Arrangements to cover expenses of entertainment, the clinic, publicity, and other features. On the other hand, these expenses will be met very largely with the increased registration fee (previously one dollar) paid by those who actually attend the convention.

All members of the A. V. M. A. in good standing (dues for 1937 paid) will pay a registration fee of $2.50 and a similar fee for each lady accompanying the member.

Non-members of the A. V. M. A. will pay a registration fee of $3.50 per person and a fee of $2.50 for each lady accompanying a non-member. No registration fee for children.

Junior members of the A. V. M. A. will be allowed to register without the payment of any registration fee, upon presentation of a membership card for 1936-1937.

The registration badge will entitle the wearer to attend all sessions of the convention, including the clinic, but not to attend any entertainment for which tickets are sold (banquet and alumni dinners).

Entertainment for Ladies

TUESDAY, AUGUST 17

Ladies will attend the opening session of the convention in the Ball Room, at 10:00 o'clock. A reception and tea at the Aquila Court has been arranged for the ladies, at 3:00 o'clock. In the evening, the ladies will attend the polo game and Den Show at the Ak-Sar-Ben Coliseum.

WEDNESDAY, AUGUST 18

At 10:30 o'clock a complimentary breakfast will be served at the Omaha Athletic Club, followed by the annual meeting of the Women's Auxiliary to the A. V. M. A. In the evening, the ladies will attend the general banquet, in the Ball Room. This will be followed by the President's Reception and Dance.

THURSDAY, AUGUST 19

At 12:30 o'clock, luncheon will be served for the ladies at the Paxton Hotel, in conjunction with a style show. Most of the

alumni groups that will have dinner meetings, at 6:00 o'clock, usually have the ladies present. Information on this point may be obtained at the Registration Bureau.

FRIDAY, AUGUST 20

The ladies will visit the Joslyn Memorial Art Museum where a special program of classical music has been arranged.

HOSTESSES AT OMAHA

Standing: Mrs. L. F. Nisley, Mrs. D. W. Hurst, Mrs. J. D. Ray, Mrs. W. T. Spencer, Mrs. E. H. Carter.

Seated: Mrs. Frank Breed, Mrs. J. A. Lueth, Mrs. A. C. Drach, Mrs. C. J. Norden, Mrs. T. W. Munce.

Polo Game and Den Show

On Tuesday evening, August 17, from 6:45 to 7:45 o'clock, a polo game will be played by the All-Stars and the Omaha Lakewood Club, at Ak-Sar-Ben Field, adjacent to the Coliseum. The All-Stars will be co-captained by Drs. G. E. Van Tuyl and A. L. Birch, practicing veterinarians of Paullina, Iowa, and Worthington, Minn. The Omaha Lakewood Club is one of the outstanding polo clubs of the Midwest. This event is sponsored by the Associated Serum Producers, Inc.

Following the polo game (at about 8:00 o'clock), a performance of the Ak-Sar-Ben Den Show will be given in the Coliseum,

adjacent to Ak-Sar-Ben Field. "Historical Hysteria," a colorful, rollicking, musical extravaganza depicts a misconception of early pioneer days; brave cowboys and soldiers, desperate outlaws, noble Indians, faro queens and colorful maidens, with a musical chorus of 100 voices, written, directed and staged by Ak-Sar-Ben talent. Interwoven into this performance are surprising initiation specialties highly entertaining to all except those initiated. It is said to be the most unusual show in America.

The Knights of Ak-Sar-Ben is on the nation's foremost civic organizations, with 6,000 members, all citizens of Omaha and Nebraska. It sponsors many community activities, including the Ak-Sar-Ben Live Stock Show and Exposition; the Ak-Sar-Ben race meet; the Coronation Ball, the outstanding society event of the Midwest, and the weekly Den Show, where thousands of Knights and visitors are entertained.

CAPTAINS OF THE ALL-STARS

Dr. G. E. Van Tuyl, of Paullina, Iowa, and Dr. A. L. Birch, of Worthington, Minn., both practitioners, will co-captain the All-Stars.

The performance of this famous Den Show for the A. V. M. A. will be provided through the courtesy of the following Omaha packing companies: Armour and Company, Cudahy Packing Company, Swift and Company; other live stock interests, and the Omaha Chamber of Commerce.

MARYLAND ACCREDITED

On July 1, Maryland was added to the list as the 44th State to be declared tuberculosis-free by the U. S. Department of Agriculture, the first state to be accredited during 1937, leaving only four states without this coveted distinction—California, New Jersey, New York and South Dakota. June was a month of marked progress in three of these states. California added four counties to the accredited list, New York did likewise with four counties, while South Dakota accredited five counties.

COLONEL AND MRS. ROBERT J. FOSTER

This photograph was not taken at Omaha, but fifteen miles north of Quito, Ecuador, on November 2, 1928. The equatorial line passes through the notch in the mountains at the right of Col. Foster. Just getting acclimated?

Meet—

HON. ROBERT FECHNER
Director, Emergency Conservation Work, Washington, D. C. Will address the general session on Tuesday afternoon.

DR. C. E. WALLER
Assistant Surgeon General, U. S. Public Health Service, Washington, D. C. Will address the general session on Tuesday afternoon.

DR. CASSIUS WAY
Chairman of the A. V. M. A. Executive Board. Will respond to the address of welcome at the opening session.

DR. J. D. SPRAGUE
Secretary of the Nebraska State Veterinary Medical Association. Joined the A. V. M. A. at the 1898 meeting in Omaha.

Exhibits at the OMAHA CONVENTION

ABBOTT LABORATORIES

North Chicago, Ill.

Abbott Laboratories, manufacturers of pharmaceutical and biological products for the veterinary profession, will display in their exhibit such outstanding items as Nembutal, Pentothal Sodium, Metaphen in Kaolin, Sulfanilamide and **Abbott** Nutritional Products, including Haliver Oil preparations and Vita-King.

ALLIED LABORATORIES—PITMAN-MOORE COMPANY

Kansas City, Mo.—Indianapolis, Ind.

The **Allied Laboratories—Pitman-Moore** exhibit will feature pharmaceutical and biological products, arranged according to animal species, and an interesting motion picture of a trip through a serum plant. Mr. F. V. Hawkins, secretary of **Pitman-Moore Company,** and several members of the **Allied** staff will be in charge.

BECTON, DICKINSON AND COMPANY

Rutherford, N. J.

Becton, Dickinson and Company will exhibit their well-known line of veterinary specialties, including Champion Syringes and Needles, Intravenous Injections, and a new Bleeding Needle Holder for taking specimens for Bang's disease testing, developed in coöperation with the Bureau of Animal Industry. Samples of a new milking-tube will be distributed.

CORN STATES SERUM COMPANY

Omaha, Neb.

The exhibit of **The Corn States Serum Company** will emphasize the serum-virus production methods and equipment in use at its establishments, together with a pleasing display of pharmaceutical, biological and sundry products for graduate veterinarians.

"There is no Substitute for Satisfaction"

is the official slogan of this progressive institution, whose home office and producing establishments are adjacent to Omaha.

CUTTER LABORATORIES

Berkeley, Calif.

The **Cutter Laboratories** will feature their latest biological specialties, including Gonadin. Of particular interest will be motion pictures of the work being done by this firm with Hog Cholera Antigen (Tissue Vaccine), developed by Dr. William Hutchins Boynton. A competent representative will be on hand to answer questions concerning it. The **Cutter** film on Equine Encephalomyelitis will be shown at regular intervals.

FORT DODGE LABORATORIES, INC.

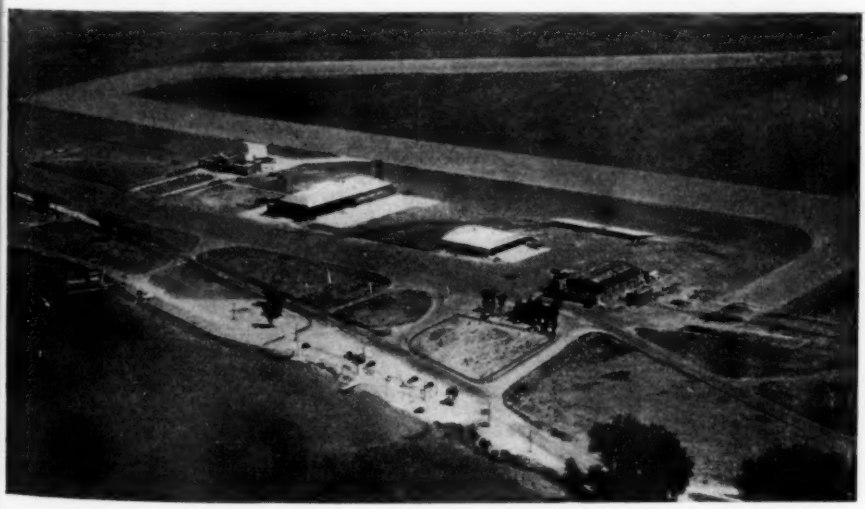
Fort Dodge, Iowa

Fort Dodge will have a most interesting and comprehensive exhibit of its up-to-date and complete line of veterinary biological and pharmaceutical products. An interesting motif of this exhibit will be the accentuation of the fact that this is the Silver Anniversary of the **Fort Dodge Serum Company.**

HARRIS X-RAY CORPORATION

Chicago, Ill.

Harris X-Ray Corporation will feature the Little Mogul Portable Shockproof X-Ray Unit for radiographic-fluoroscopic work, for extemporaneous use in general radiographic work, bone-setting and other essential operative procedure. Also specimen radiographs and photos, showing the x-ray machine operating in unusual situations, will be on display.



OMAHA MUNICIPAL AIRPORT

HAVER-GLOVER LABORATORIES

Kansas City, Mo.

This exhibit will display a map of the United States, with intermitten lights depicting the Coast-to-Coast service depots of the **Haver-Glover Laboratories.** Space in this elaborate background is provided for certain featured pharmaceutical and biological products, and in the foreground the usual complete display of surgical requirements.

JENSEN-SALSBERY LABORATORIES, INC.

Kansas City, Mo.

The **Jensen-Salsbery Laboratories** will display a full line of veterinary instruments, and pharmaceutical and biological products under the popular trade-mark **Jen-Sal.** Most of the products offered by this company have been developed in the field of clinical medicine, which has made notable advancements in the past few years. The display will effectively reflect the advancement in equipment which has been made.

LEDERLE LABORATORIES, INC.

New York, N. Y.

Lederle Laboratories, Inc., will have biological and pharmaceutical products on display. There will be a special featuring of Anti-Hog Cholera Serum and Virus, Canine Distemper products, and the new Black Tongue Specific for dogs. Our local representatives will be on hand to greet you; also members of the technical staff from New York.

ASHE LOCKHART, INC.

Kansas City, Mo.

This exhibit will display the fifty-odd biological products prepared by **Ashe Lockhart, Inc.,** for the use of graduate veterinarians. Diagnostic agents, serums, vaccines, bacterins, aggressins, antitoxins, tissue extracts. Printed matter which we have prepared for veterinarians to distribute to their clientele, emphasizing several phases of veterinary service, will also be available.

NORDEN LABORATORIES

Lincoln, Neb.

The Most interesting feature of the **Norden** exhibit will consist of a motor-driven tablet machine in operation, making Sulfamide tablets. In addition, there will be a number of other products, such as Certified Brand anti-hog cholera serum and a few selected pharmaceuticals, including Norcalcinate products. Look for the **Viking Ship.**

RIVAL PACKING COMPANY

Chicago, Ill.

The **Rival Dog Food** exhibit will be one of beauty and attractiveness. While the display is not technical, members of the Rival organization who will be in charge of the exhibit are well qualified to demonstrate to visiting veterinarians the superior claims that the **Rival Packing Company** makes for its product, Rival Dog Food.

SPICER AND COMPANY

Glendale, Calif.

Spicer and Company offer ten new products representing recent advances in immunotherapy and organotherapy in veterinary medicine. Facilities have been developed to perfect means whereby newer antibacterial knowledge may be applied in clinical practice. Today's weapons against infection and its resutls on the organism represent some of the greatest triumphs in medical research.

R. J. STRASENBURGH COMPANY

Rochester, N. Y.

Several new **Strasenburgh** products will be exhibited under the direction of Mr. W. J. Smith, assistant sales manager. Sulfanilamide (in tablets), Alopectose and Ven-apis are among the outstanding new preparations which will be shown. Complete information and leaflets will be available to those interested. 1937 marks the 51st year of this firm.

SWIFT AND COMPANY

Chicago, Ill.

The Pard exhibit of **Swift and Company,** for the 74th annual convention of the American Veterinary Medical Association in Omaha, will feature a pictorial presentation of up-to-date information regarding canine nutrition and an actual demonstration, which will be of interest to all veterinarians.

WILSON AND COMPANY

Chicago, Ill.

Wilson and Company will display Ideal Dog Food and Old Trusty Dry Dog Foods. Growth charts, based on feeding experiments conducted by a leading midwestern university, will show the efficiency of Ideal Dog Food as a ration for dogs, chicks and rats. Visitors will receive a complimentary booklet containing illustrated descriptions of the 34 most popular breeds of dogs.

EXHIBIT FUND GROWING

Ten more state associations have made appropriations to the fund being raised by the A. V. M. A. for the purpose of financing educational exhibits at the New York World's Fair and the San Francisco Exposition in 1939. The total number of states that have made appropriations is now 18, and the grand total of these is $1,700.00.

The ten additional amounts are:

New York State Veterinary Medical Society	$ 500.00
Illinois State Veterinary Medical Associatnon	200.00
California State Veterinary Medical Association	100.00
Oklahoma Veterinary Medical Association	100.00
Connecticut Veterinary Medical Association	50.00
Nebraska State Veterinary Medical Association	50.00
Mississippi State Veterinary Medical Association	25.00
North Dakota Veterinary Association	25.00
Virginia State Veterinary Medical Association	25.00
Wyoming Veterinary Association	20.00
Previously acknowledged (See May JOURNAL)	605.00
	$1,700.00

If any other state association, not mentioned in either of these reports, has made an appropriation, the secretary should notify the secretary of the A. V. M. A. just as soon as possible. Several associations have given serious consideration to the matter, but on account of the depleted condition of their treasuries have not felt that appropriations could be made at this time.

APPLICATIONS FOR MEMBERSHIP

(See July, 1937, JOURNAL)

FIRST LISTING

ANDERSEN, ANDREW C. P. — Centerville, Iowa
D. V. M., Iowa State College, 1933
Vouchers: F. F. Meads and C. R. Fry.

BAKER, JACK E. — 717 Penrith Dr., Los Angeles, Calif.
B. S., University of California, 1929
D. V. M., Cornell University, 1937
Vouchers: Arnold S. Rosenwald and C. A. White.

BALDWIN, CHARLES R. — Fulton, N. Y.
D. V. M., Cornell University, 1906
Vouchers: Cassius Way and W. A. Hagan.

BRADLEY, ALFRED M. — Alvord, Iowa
D. V. M., Indiana Veterinary College, 1921
Vouchers: T. W. Munce and N. L. Nelson.

BROWN, WILLIAM E. — Ankeny, Iowa
D. V. M., Iowa State College, 1937
Vouchers: C. H. Covault and Chas. Murray.

CARDONA, CARLOS J. Escuela Practica de Agricultura, Maracay, Venezuela
D. V. M., Texas A. & M. College, 1936
Vouchers: H. L. Van Volkenberg and H. V. Cardona.

CARPENTER, ERNEST R. Minnesota Lake, Minn.
B. V. Sc., Ontario Veterinary College, 1924
Vouchers: Joab P. Foster and Carl Hansen.

CARTER, EARL H. 203 Federal Bldg., South Omaha, Neb.
V. S., Ontario Veterinary College, 1906
M. D. V., McKillip Veterinary College, 1907
Vouchers: W. T. Spencer and J. D. Ray.

CORWIN, THOMAS E. 8701-80th St., Woodhaven, L. I., N. Y.
D. V. S., New York-American Veterinary College, 1907
Vouchers: Harry B. Risley and R. W. Gannett.

CRANDALL, JAMES C. 163 Ulster Ave., Saugerties, N. Y.
D. V. M., Cornell University, 1931
Vouchers: Arthur L. Smith and Clifford H. Milks.

DAVIDSON, GRAHAM R. Box 168, La Grande, Ore.
D. V. M., Colorado State College, 1935
Vouchers: F. M. Bolin and B. T. Simms.

DERMODY, THOMAS A. Breda, Iowa
D. V. M., Iowa State College, 1932
Vouchers: Amor C. Drach and T. J. Beirne.

EMPEY, WILLIAM H. Battle Creek, Iowa
D. V. M., Kansas City Veterinary College, 1916
Vouchers: A. H. Quin, Jr., and T. W. Munce.

FAILING, GEORGE S. Winona, Minn.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: Joab P. Foster and John S. Dick, Jr.

FRANCESILLA, JOHN R. 315 W. Lancaster Ave., Downingtown, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and J. V. McCahon.

FREY, CHARLES J. 8109 Georgia Ave., Silver Spring, Md.
D. V. S., United States College of Veterinary Surgeons, 1906
Vouchers: W. E. Cotton and John R. Mohler.

FRIEZE, HARRY L. Gaffney, S. C.
D. V. M., Colorado State College, 1920
Vouchers: M. R. Blackstock and W. A. Barnette.

GIBBONS, JAMES L. 1372 Market St., Chehalis, Wash.
D. V. M., Iowa State College, 1936
Vouchers: V. C. Pauhlman and P. G. MacKintosh.

GINN, WILLIAM 409 State Office Bldg., Columbia, S. C.
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: M. R. Blackstock and R. A. Mays.

GLASCOCK, DALE WM. Danbury, Iowa
D. V. M., Iowa State College, 1928
Vouchers: A. H. Quin, Jr., and T. W. Munce.

HANER, FRANK H. Hensonville, N. Y.
D. V. M., Cornell University, 1914
Vouchers: Wm. Henry Kelly and Cassius Way.

HAYES, WINTHROP L. Neola, Iowa
D. V. M., Iowa State College, 1921
Vouchers: Amor C. Drach and T. J. Beirne.

HEIN, HUBERT R. Greenleaf, Kan.
D. V. M., Kansas State College, 1935
Vouchers: R. W. Johnson and J. C. Luckeroth.

HELMS, ORLAND E. East Randolph, N. Y.
D. V. M., Cornell University, 1920
Vouchers: Cassius Way and C. P. Zepp.

HOFFMANN, LEONARD F. 1131 E. Lincoln, Estherville, Iowa
D. V. M., Iowa State College, 1932
Vouchers: T. W. Munce and N. L. Nelson.

HORSTMAN, ARTHUR W. 1741 W. Main Ave., Houston, Texas
D. V. S., Kansas City Veterinary College, 1909
Vouchers: Howard L. Darby and Matthew E. Gleason.

HUGHES, EMORY G. Sleepy Eye, Minn.
D. V. M., Iowa State College, 1934
Vouchers: N. L. Nelson and L. E. Stanton.

HUNT, CLARENCE E. 211 E. Monroe St., Mount Pleasant, Iowa
D. V. M., Iowa State College, 1908
Vouchers: P. V. Neuzil and T. W. Munce.

JACOBS, O'NEAL Laurens, S. C.
D. V. M., Ohio State University, 1920
Vouchers: W. A. Barnette and M. R. Blackstock.

JACOBS, THOMAS BOYD Newberry, S. C.
D. V. M., Ohio State University, 1914
Vouchers: M. R. Blackstock and W. A. Barnette.

JAGGI, F. P. 860 Fannin, Beaumont, Texas
D. V. M., Texas A. & M. College, 1926
Vouchers: J. G. Kerr and P. W. Burns.

JENNINGS, MICHAEL T. Corning, Iowa
D. V. M., Saint Joseph Veterinary College, 1920
Vouchers: Paul L. Matthews and George Wessels.

JENSEN, J. A. Marshalltown, Iowa
D. V. M., McKillip Veterinary College, 1916
Vouchers: T. W. Munce and E. A. Cahill.

JOEHNK, ALFRED H. 732 E. Jefferson St., Iowa City, Iowa
M. D. C., Chicago Veterinary College, 1908
Vouchers: J. H. Spence and C. E. Juhl.

JOHNSTONE, JAMES T. 309 Federal Bldg., Pierre, S. Dak.
D. V. M., Ohio State University, 1936
Vouchers: G. E. Melody and C. H. Hays.

JONES, GEORGE S. 409 State Office Bldg., Columbia, S. C.
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: M. R. Blackstock and R. A. Mays.

JORGENSEN, JAMES P. Elkhorn, Iowa
D. V. M., Iowa State College, 1906
Vouchers: Amor C. Drach, W. T. Spencer and R. L. South.

KAHLE, ERNEST C. Le Center, Minn.
D. V. M., McKillip Veterinary College, 1916
Vouchers: Joab P. Foster and William C. Prouse.

KAPLUS, ELLIOT 787 Clinton Ave., Newark, N. J.
D. V. M., Alabama Polytechnic Institute, 1937
Vouchers: Don A. Yandell and I. S. McAdory.

KENNEDY, CHARLES E. 403 Saint Anthony St., Mobile, Ala.
D. V. M., Alabama Polytechnic Institute, 1932
Vouchers: Lt. Houston Odom and I. S. McAdory.

KITCHEN, DONALD O. Greenville, S. C.
D. V. M., Iowa State College, 1933
Vouchers: M. R. Blackstock and Frank Kitchen.

KNOOP, FRED W. LeMars, Iowa
D. V. M., Indiana Veterinary College, 1909
Vouchers: G. B. Fincham and T. W. Munce.

LAWRENCE, CHARLES C. Manilla, Iowa
D. V. M., Iowa State College, 1927
Vouchers: T. W. Munce and J. D. Thrower.

LINDSEY, MARK B. 1911 S. Main St., Santa Ana, Calif.
D. V. M., Colorado State College, 1934
Vouchers: W. L. Curtis and G. C. Green.

MAJERUS, CARL J. Falls City, Neb.
D. V. M., Kansas State College, 1931
Vouchers: J. E. Weinman and Amor C. Drach.

MCCUTCHAN, HOWARD E. Ayrshire, Iowa
D. V. M., Iowa State College, 1936
Vouchers: H. D. Bergman and N. L. Nelson.

MOORE WILLIAM M. Westmoreland Farm, Route 1, Cary, N. C.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. A. Husman and Wm. Moore.

MOTTRAM, WILLIAM E. 355 Fair Oaks St., San Francisco, Calif.
D. V. M., State College of Washington, 1937
Vouchers: C. B. Miller and M. J. O'Rourke.

NELSON, WALTER I. Herman, Neb.
D. V. M., Saint Joseph Veterinary College, 1923
Vouchers: G. J. Collins and Paul L. Matthews.

RAINEY, PAUL A. Box 6123, West Palm Beach, Fla.
D. V. M., Ohio State University, 1936
Vouchers: T. H. Applewhite and B. N. Lauderdale.

RASMUSSEN, VICTOR M. Sleepy Eye, Minn.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: W. A. Anderson and L. S. Englerth.

REID, GEORGE F. 832 Washington St., Albany, Ore.
V. S., B. V. Sc., Ontario Veterinary College, 1921
Vouchers: Geo. D. Bishop and B. T. Simms.

RESSEGUIE, ROY A. Charter Oak, Iowa
D. V. M., Iowa State College, 1936
Vouchers: A. H. Quin, Jr., and E. E. Grove.

SCHWAB, JOHN T. 403 W. Wisconsin Ave., Oconomowoc, Wis.
D. V. M., Saint Joseph Veterinary College, 1923
Vouchers: Herbert Lothe and James S. Healy.

SCHOLTY, W. C. Leon, Iowa
D. V. M., Iowa State College, 1902
Vouchers: Amor C. Drach and T. J. Beirne.

SHARP, FLOYD S. Ute, Iowa
D. V. M., Kansas City Veterinary College, 1916
Vouchers: T. W. Munce and N. L. Nelson.

SHINDELL, SAMUEL H. 164 E. 71st St., New York, N. Y.
D. V. M., Cornell University, 1916
Vouchers: E. Sunderville and C. P. Zepp.

SIBERT, HERBERT F.
Hdqrs. Missouri-Kansas CCC, Fort Leavenworth, Kan.
D. V. M., Kansas State College, 1934
Vouchers: Lt. Col. Jesse D. Derrick and Lt. H. L. Morrison.

SINNERUD, OTTO P. 10418 Vincennes Ave., Chicago, Ill.
D. V. M., Chicago Veterinary College, 1920
Vouchers: H. Preston Hoskins and John B. Jaffray.

STOREY, ENSLY R. Box 97, Albany, Ore.
B. S., D. V. M., State College of Washington, 1935
Vouchers: Geo. D. Bishop and L. R. Libby.

THERKELSEN, FRANK W. Jeffers, Minn.
D. V. M., Chicago Veterinary College, 1916
Vouchers: Louis E. Stanton and C. P. Fitch.

THIELE, ARTHUR R. 408-410 State Office Bldg., Columbia, S. C.
D. V. M., Kansas State College, 1934
Vouchers: Edwin J. Frick and W. K. Lewis.

VAN DER MAATEN, MARTIN. Alton, Iowa
D. V. M., Kansas State College, 1929
Vouchers: A. H. Quin, Jr., and T. W. Munce.

VAN TUYL, GEORGE E. Paullina, Iowa
D. V. M., Chicago Veterinary College, 1917
Vouchers: G. B. Fincham and T. W. Munce.

VERNON, JOHN M. 636 Des Moines St., Des Moines, Iowa
M. D. C., Chicago Veterinary College, 1908
Vouchers: A. H. Quin, Jr., and E. E. Grove.

WEISHEIT, HENRY C. Glenmont, N. Y.
D. V. M., Cornell University, 1935
Vouchers: J. G. Wills and Charles Linch.

Applications Pending

SECOND LISTING

(See July, 1937, JOURNAL)

Ahlers, Fred R., La Motte, Iowa.
Anderson, Alfred A., 518 W. 3rd St., Grand Island, Neb.
Anderson, Thomas E., Bedford, Iowa.
Andrews, William L., Milton, Iowa.
Armstrong, James W., 95 Eldridge St., Cranston, R. I.
Armstrong, William H., 601 S. Elena Ave., Redondo Beach, Calif.
Baer, J. W., Malvern, Iowa.
Baird, Norman G., Sanford Veterinary Hospital, Sanford, N. C.
Bartenslager, Arthur V., Stewartstown, Pa.
Beard, R. V., 2535 Mount Pleasant Rd., Burlington, Iowa.
Blank, Lloyd C., Emerson, Iowa.
Bowstead, Leonard A., Dewitt, Iowa.
Boyle, William H., 932 Cochran Ave., Schuyler, Neb.
Brady, Ezra M., 708 E. 9th St., Atlantic, Iowa.
Bromwell, V. G., Center Point, Iowa.
Buchleiter, Walter H., Coin, Iowa.
Buckley, Howard P., c/o Dr. G. S. McKee, Beaver Falls, Eastvale, Pa.
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The amount which should accompany an application filed this month is $7.08, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

San Diego County Veterinary Medical Association. San Diego, Calif. August 10, 1937. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

Poultry Science Association. University of Wisconsin, Madison, Wis. August 10-13, 1937.

American Veterinary Medical Association. Hotel Fontenelle, Omaha, Neb. August 16-20, 1937. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. August 18, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

American Animal Hospital Association. Hotel Fontenelle, Omaha, Neb. August 19, 1937. Dr. A. D. Eastman, Secretary, 901 Nineteenth St., Moline, Ill.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. September 1, 1937. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. September 1, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. September 2, 1937. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. September 4, 1937. Dr. Robert L. Hummer, Secretary, 312 W. Church Av., Knoxville, Tenn.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. September 7, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. September 8, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. September 13, 1937. Dr. W. H. Riser, Secretary, Glenwood, Iowa.

Chicago Veterinary Medical Association. Anti-Cruelty Society, 157 W. Grand Ave., Chicago, Ill. September 14, 1937. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Willamette Valley Veterinary Medical Association. Chamber of Commerce Rooms, Salem, Ore. September 15, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Southwestern Minnesota Veterinary Medical Association. Luverne, Minn. September 21, 1937. Dr. Louis E. Stanton, Secretary, Jackson, Minn.

Veterinary Association of Saskatchewan. University of Saskatchewan, Saskatoon, Sask. October 1, 1937. Dr. Norman Wright, Secretary, 137 20th St. West, Saskatoon, Sask.

New Mexico Veterinary Medical Association. Albuquerque, N. M. October 1-2, 1937. Dr. T. I. Means, Secretary, Penn Rd., Santa Fe, N. M.

New England Veterinary Medical Association. Portland, Me. October 4-5, 1937. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

American Humane Association. Milwaukee, Wis. October 4-7, 1937. Mr. N. J. Walker, Secretary and General Manager, 80 Howard St., Albany, N. Y.

International Association of Milk Sanitarians. Brown Hotel, Louisville, Ky. October 11-13, 1937. Mr. C. Sidney Leete, Secretary, Department of Health, Albany, N. Y.

Southern States Veterinary Medical Association. Ansley Hotel, Atlanta, Ga. October 14-16, 1937. Dr. M. R. Blackstock, Secretary, 157 W. Hampton Ave., Spartanburg, S. C.

Purdue University Veterinary Short Course. Purdue University, Lafayette, Ind. October 19-22, 1937. Dr. R. A. Craig, Department of Veterinary Science, Purdue University, Lafayette, Ind.

West Virginia Veterinary Medical Association. Fairmount Hotel, Fairmount, W. Va. October 25-26, 1937. Dr. J. H. Rietz, Secretary, Oglebay Hall, West Virginia University, Morgantown, W. Va.

Hog Slaughter Takes Big Drop

During the month of May, 1937, the number of hogs slaughtered was the smallest for any May since 1895. There were only 2,098,590 hogs killed, compared with 2,809,788, in April, and 2,579,450 in May, 1936. Four years ago, before the drouths and federal control of production, the May slaughter of hogs totaled 4,286,239, more than double the number killed in May this year. Pork in storage decreased 92,400,000 pounds during May.

FOWL LEUKOSIS*

By C. D. LEE, H. L. WILCKE, CHAS. MURRAY *and* E. W. HENDERSON

Iowa State College, Ames, Iowa

During recent years, a great deal of work has been done to determine the true nature of fowl leukosis, especially from the standpoint of its etiology and the mode of transmission. A disease if such great economic importance and of such biologic interest invited further study and the investigations begun at Iowa State College, in February, 1930, are still in progress.

In the beginning we were primarily concerned with manifestations of paralysis and blindness, but early in our studies we were impressed by the frequent occurrence of tumors and tumor-like infiltrations in various organs and tissues of the so-called paralysis cases. Certain histologic structures, varying from slight cellular infiltration to hyperplasia and tumors of the same type of cells, lead one to conclude that paralysis is only one symptom associated with the malady. Later, the close association of leukemic-like and erythroleukosis-like manifestations became very apparent, thus making a study of these various expressions seem imperative.

The complexity of the disease has led to a variable nomenclature, which expresses in most cases a quite adequate nomenclature for a single manifestation of the disease but inadequate as a name for this disease as a whole. There is usually considerable overlapping in the various expressions of the disease, so that in each case the nomenclature used pertains to the type of cells in predominance. It would appear that all types come from undifferentiated cells that cannot be differentiated as long as they remain in the undifferentiated blast stage. These cells are the predominating cells in the lesions and are termed hemocytoblasts which are cells possessing potentialities toward any of the blood-cell types.

A particular case in question can be definitely classified into a definite type only when enough of these cells have been differentiated sufficiently to determine their developmental tendencies. Fowl leukosis is believed to be a more apt term and may be used to designate in common all forms of the disease. Fowl leukosis is most commonly differentiated into three types, erythroid, myeloid and lymphoid, and may be further differentiated as nerve

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type, eye type and visceral lymphocytoma, according to site of lesion.

Time does not permit a complete review of the literature but a review of the literature shows there are marked differences of opinion in regard to the relationship of the various manifestations in this condition. It is generally agreed that myeloid leukosis and erythroleukosis are types of the same transmissible disease. It is also quite generally agreed that the nerve type (neuro-lymphomatosis gallinarum) and the eye type are closely related. There is, however, a difference of opinion regarding the relationship of neurolymphomatosis gallinarum and lymphoid leukosis. There are also marked differences of opinion regarding the relationship of erythroleukosis and myeloid leukosis and lymphoid leukosis. It seems quite generally agreed that the etiological agent is a filtrable virus.

Clinical Symptoms

The clinical symptoms in this disease complex vary according to the location and degree of involvement of the affected tissues or organs. Cases suffering from lymphoid leukosis or hemocytoblastosis of the nerve type may be observed as a unilateral, bilateral, local or general paralysis. Paralysis of one or both legs or paralysis of one or both wings is most commonly observed. These manifestations may be only slight or may be so severe that the fowl is practically helpless. Frequently, when the legs are involved, common characteristics may be observed, such as stiffness, lameness, walking in a stilty manner with a tendency toward extending the affected leg or legs backward, forward or straight from the body or one leg may be extended forward while the other is extended backward. In more advanced cases the bird may go completely down, dragging itself about assisted by the wings.

When the brain is involved, various lethargic symptoms are observed as well as abnormal head movements, such as a weaving motion of the head, throwing the head backward or the beak downward, lateral deviations and in general a lack of ability to hold the head in a normal position. Many cases show a paralysis, partial or complete, of many of the visceral organs, the severity depending upon the amount of involvement or site of involvement of nerves or nerve plexuses supplying the part.

In some cases associated with involvement of the vagus nerve, difficult respiration evidenced by breathing through the mouth has been observed. In general, any variation may be observed depending upon the nerve or nerve plexuses involved. The onset

may be gradual or sudden and the course is variable. The disease may be rapidly progressive or may progress slowly. We have never observed a complete recovery of affected cases, although temporary respites sometimes occur.

Emaciated conditions are frequently observed, especially in chronic cases. This emaciation seems to be due largely to difficulty in securing food or difficulty in utilizing it. Many cases retain their appetite throughout the course of the disease and many birds that are practically helpless will eat and drink as long as they can reach the food and water containers.

In the eye cases the iris frequently shows in the beginning a depigmented circle around the pupil. This circle is gray at first or may be only a slightly faded condition and may extend to such an extent that the normal bay color is almost completely replaced. The process may start in any part of the iris as a small depigmented spot or it may start as a generalized depigmented process throughout the iris characterized by a faded gray appearance. In many cases the depigmentation appears to start in several places in the iris at the same time, giving the appearance of areas of a normal bay color, interspersed with faded or gray areas.

In many cases the depigmentation is quite irregular and has a streaked appearance mingling the normal bay color with that of a pathological gray. The color changes may vary from a faded bay to a dark or light gray, bluish gray or slate color. In some cases a black colored tissue appears as a multiple tumorous mass. As a rule both eyes are involved, with one appearing to be more severely affected than the other. However, many cases show involvement of only one eye. In a high percentage of cases regular or irregular constriction of the pupil occurs with impaired vision or even blindness. Opacity of the cornea has been observed in a few cases.

The eye type as well as other types of fowl leukosis have a very variable course. The process may spread rapidly or very slowly and in many cases it appears as though the disease has become arrested. The birds usually remain in good health unless blindness occurs or unless eye-type cases become complicated with other expressions of the disease. Frequently we have observed cases that were uncomplicated eye cases in the beginning develop some other type after a variable length of time. We have also seen several uncomplicated eye cases that have lived two years without any great impairment of health.

While most of the popular breeds of chickens should have normally a reddish bay-colored iris, there are some that do not have.

The iris of the Cornish, and some game birds, is normally a yellow or pearl color and many crosses of this breed may have normally pearl or gray eyes. Some breeds also have normally a dark brown or nearly a mahogany-colored iris. Some strains of breeds that are supposed to have reddish bay irises may produce many individuals that have light-colored irises. The above factors, as well as faded or gray irises from other causes, such, as general weakness, different feeding practices and dietary factors, should always be considered in making diagnoses in questionable cases.

Many degrees of clinical manifestations are noted in cases of the greatly varying erythroid type. The most characteristic symptom shown is a severe anemia with a definite icterus of the skin of the face. The tendency in most of these cases is toward emaciation. However, the condition of flesh in many cases may be deceiving, as it is frequently observed that birds appearing to be in excellent flesh are somewhat edematous and soft. Positive blood findings in the peripheral circulation of numerous hemocytoblasts and basophil erythroblasts, presumably precursors of erythrocytes, are frequently encountered and a negative blood-picture may not at all times mean that the bird is not affected with some expression of the disease. The course of the disease is also very variable, in some cases progressing rapidly and in others slowly. The disease may appear to be arrested, but complete recovery has never been noted. The severity of symptoms is no indication of the extent of involvement. The appetite usually remains good except in the very acute, generalized case and in cases of prostrated birds.

The clinical manifestations of myeloid leukosis are very variable. The onset may be gradual or very rapid and general weakness with diarrhea is quite common. Marked lameness is frequently found in those cases where marked infiltration in various organs is found on autopsy. The lameness may occur in one or in both legs, but in most cases occurs in both. The skin, comb and wattles may not show any color changes, while in some cases varying degrees of anemia as well as a peculiar pallor may be observed. The presence of large numbers of myelocytic elements in the peripheral circulation serves to make a diagnosis. The tendency in these cases is toward a rather rapid emaciation.

Clinical symptoms in cases affected with hemocytoblastoma and lymphocytoma tumors and tumor-like infiltrations in various organs vary greatly, depending upon the tissues or organs involved. Lameness has been observed in cases where a tumorous mass was found pressing on a corresponding nerve or nerve

plexus. Diarrhea and emaciation are quite common in the more chronic cases. Many cases in good flesh have died suddenly and upon autopsy were found to be suffering from an enormously enlarged, infiltrated liver. Rupture of the liver has occurred frequently in such cases. Pendulous abdomen has been observed in a few cases. Respiratory difficulty was observed in a few cases which, upon autopsy, showed an involvement of the lungs by lymphocytomatosis and hemocytoblastosis tissue infiltrations. The symptoms in general are more obscure than in the other types of the disease. A negative blood-picture does not necessarily mean the bird is not affected.

PATHOLOGY

Nerve lesions: In the nerve type the most characteristic lesions are enlargement of nerves or nodular formation in nerves with hemocytoblasts and lymphocyte infiltrations. In many cases the nerves appear only slightly swollen and edematous, while in others the enlargements may be extreme. The white, glistening appearance and irregular striations such as are common to normal nerves are usually less distinct in mild cases and completely disappear from those in badly affected cases. The infiltrated nerve is soft, tumor-like, and a dull gray or fatty yellow in color. The process may be confined to only one nerve or may affect a group of nerves or it may be more generalized, affecting many groups of nerves. The most severe lesions were those observed in the peripheral nerves and the most common sites were the ischiatic, vagus, brachial, lumbosacral, splanchnic and intercoastal plexuses and their branches. The nerve roots and ganglion tissue along the spinal column are very often involved.

The pathologic manifestations may be slight and consist essentially of an infiltration of small, intermediate, or large mononuclear cells between the nerve fibers, separating them and accompanied by more or less edema. In many cases the invading cells are loosely distributed in certain areas of the nerve. In more severe manifestations the invading tissue may almost completely replace the nerve tissue of a nerve or plexus. The tendency is toward a diffuse type of infiltration, although great variation may be observed in different areas of the same nerve. Histologic studies usually reveal an infiltration of variable sized, undifferentiated, mononuclear, basic staining, non-granular cells that are considered on the same basis as undifferentiated embryonic blood progenitors (hemocytoblasts). They are apparently the same type cells as those usually called lymphoid-like, and have the same characteristics and variations as those found

in aleukemic and leukemic lymphocytoma, large or small round cell sarcoma and lymphosarcoma. Mitotic figures are frequently observed in cells of the nerve lesions, especially in the intermediate type cells.

Degeneration changes in the nerve fibers vary greatly in the different cases and the extent of degeneration present depends on the amount and intensity of the infiltration.

Spinal-cord lesions: The lesions in the central nervous system are most often found by histologic examination. In a few cases gross lesions of extensive proportion are observed in the brain and the spinal cord. Many of the lesions in the spinal cord originate in the peripheral nerves and extend to the spinal cord. However, this is not always true, as many cases are found with lesions in the central nervous system with apparently no other nerve tissue involved. In the spinal cord, in addition to the infiltrations accompanying the entrance of a nerve-root, there are found focal accumulations of cells more often in the white matter but in some instances also in the central gray. Lesions of this type seem to be limited to the vicinity of the smaller blood-vessels. The cells composing these accumulations appear to be hemocytoblasts and lymphocytes. Degeneration changes in the ganglion cells of the cord are observed only when the lymphoid-like infiltration is so massive as to bring about extensive destruction of the cord tissue.

Brain lesions: The lesions found in the brain are always focal. They are frequently found in the cerebrum, cerebellum, optic thalmus and pons. With one exception they were always perivascular rings of compact, densely staining, small lymphocytes. In only one case was a diffuse infiltration, as observed in nerves and the spinal cord, found and this case was characterized by a definite, extensive, tumor-like, gross lesion.

Eye lesions: The lesions found in the iris and pupil are usually less distinct after death than before. This makes it very desirable to examine the eyes before death whenever possible. In general, the gross lesions in the iris and pupil are not greatly different from those already described under clinical symptoms. The histologic studies reveal that the iris is thickened, edematous, and infiltrated with the same type of blast-stage cells as described in nerve lesions. These appear as small and large, basic staining, non-granular hemocytoblasts and lymphocytes. The larger elements frequently appear as compact areas of cells situated toward the anterior surface.

In some eye cases eosinophilic cells seem to be the predominant type, while in others there seems to be a mixture of the two types

of cells. The cornea and retina seem to have undergone no change and are regarded as normal. The optic nerve is frequently found to be involved with the same type of cells as described in other nerve-lesion cases. The conjunctiva and extraocular muscles contain dense nests of lymphoid-like cells in a few cases. The amount of infiltration in the eye cases varies greatly from slight in some cases to massive in others. There seems to be more correlation between the intensity of symptoms and lesions in this type of the disease than in the other types.

Lesions in other tissues: The lesions in some cases are confined to a single organ or tissue while in others the process is more or less generalized. However, leukemic, and aleukemic, lymphocyte-like tumors, and tumor-like infiltrations have been found in practically all of the organs and tissues of the body, the principal locations being in the liver, ovary, spleen and kidneys. They appear in some cases as diffuse infiltrations; in others, as distinct tumors. Various degrees of involvement have been observed, from microscopic foci to those of great proportions. Multiple tumors are frequently found in the liver. This tumorous tissue, which is a grayish pink or white with occasional yellow coloring, is very friable and well embedded in the affected organ. These tumors vary greatly in size from miliary foci to rather extensive formations. In many cases the infiltration is diffuse, causing a slightly or enormously enlarged liver, depending upon the extent of infiltration. The gross appearance of the liver is greatly altered, with a gray color and a roughened, hobnailed surface and rounded borders. In many cases where death occurred very suddenly, it was found that the abdominal cavity was filled with blood due to rupture of a very friable liver.

The spleen and kidneys are affected much as the liver and in many cases all three organs show changes. The ovary is very frequently the site of infiltrations. It may be enlarged several times its normal size, and its normal structure may be obliterated by a mass of soft, yellowish-gray, infiltrating, lymphomatous tissue. In one case, with a large tumorous mass in the ovary, there was a direct extension to the kidney. In another case, an extension from an affected ovary directly into the spinal canal was observed, with lesions in the cord identical with those found in the cord of the nerve type. Histopathologic studies reveal the same type of undifferentiated cells as found in nerve lesions. In the liver the infiltrations vary from mild periportal accumulations to massive infiltrations, which so destroy the liver cells that in many cases it is impossible to demonstrate the normal liver tissue.

In the kidneys the condition is analogous to that in the liver. In extreme cases the infiltration found between the tubules is so great that the parenchymal elements are widely separated or actually replaced. In the spleen the infiltration is found in both the pulp and the follicles.

The tumor-like lesions found in various organs are apparently identical in their gross and microscopic appearance to those manifestations commonly described as round cell sarcoma, lymphosarcoma, leukemic and aleukemic lymphocytoma. The tedency in all cases regardless of the location of the lesion is for the pathologic cells to invade and replace the involved tissues. In many cases, histologic examination of some sections indicates that the walls of some blood-vessels and capillaries are apparently made up of invading cells.

The classifications of erythroleukosis and myeloid leukosis are based upon blood findings. In some cases with definite blood findings of erythroleukosis lesions were observed in the various visceral organs. In some cases the liver appeared swollen, with rounded edges, and the spleen enlarged and more gray in color than normal. In many cases tumor-like nodules and diffuse infiltrations of these organs occurred. The bone-marrow is always involved in erythroleukosis, being swollen, pale gray or deep red, and more solid in consistency. Microscopically an outstanding feature of erythroleukosis is the stasis of hemocytoblastic cells in the capillaries. The liver shows at times a great accumulation of these cells in the capillaries, usually intravascular but at times with tumor-like proportions. Very frequently perivascular accumulations and infiltrations occur. The spleen shows an accumulation of similar cells in the pulp and follicles. The bone-marrow trabeculae show numerous lymphocytic and myelocytic foci and in more advanced cases the entire marrow constituents appear as a mass of lymphoid-like cells.

Myeloid leukosis is characterized by the presence of myelocytes in large numbers in the peripheral blood. In some cases, diffuse or nodular infiltrations of soft, lossely arranged, gray or white myelocytomatous tissue occur in the liver, spleen, kidneys and bone-marrow.

There are also cases of myeloid leukosis, as diagnosed by blood findings, that show a definite generalized myelocytoma with tumorous formation in the liver, spleen and kidneys. Myeloid leukosis apparently begins with a tumor-like proliferation of myeloblastic and myelocytic elements in the bone-marrow, thus flooding the blood with large numbers of myeloid forms, the

termination being an accumulation of myeloid cells in the liver spleen and kidneys.

Blood and bone-marrow: The positive blood expressions seem to be largely dependent upon the involvement of the bone-marrow. When the bone-marrow is involved, expressions of myelogenous leukemia or myeloid leukosis and erythroleukosis appear in the peripheral blood. Eye and nerve types, when unaccompanied with bone-marrow changes, do not show any changes in the blood but when accompanied by lesions of this disease in the bone-marrow, may show positive blood findings.

This description has not included all the variations and mixed forms encountered in the study. The organs mentioned as sites of lesions are only a few of those that have been observed to be most frequently affected. It is safe to say that practically any organ or tissue may show lesions of this disease.

TRANSMISSION EXPERIMENTS

In conducting transmission experiments with a disease such as fowl leukosis, it seems of prime importance that one has definite knowledge of the origin of the birds used for injection. The birds used in these experiments were hatched at the laboratory from our own breeding flocks, kept solely for this purpose. A definite mating and hatching record was obtained for each chick. The chicks from each hen were separated into a series, that is, one chick for injection, one for contact and one for a control. In the event any cases of leukosis developed in the breeding flock during the experiment, the chicks from this hen and mating were immediately removed from the experiment and are not tabulated in the results. The controls were kept on a separate part of the farm and cared for by a caretaker who had no contact with other chickens. Three breeds of chickens were used—Single Comb White Leghorns, Barred Rocks and Rhode Island Reds. The Leghorns were designated as susceptible and resistant, since we believed there was a distinct difference in the susceptibility and resistance of this flock. The birds were taken directly from the incubator to the brooder houses which were equipped with screen porches and at not time were birds allowed to be on the ground.

EXPERIMENT I

Transmission by Injection and Pen Contact

A saline suspension made from an infiltrated liver of a hen that showed no eye or nerve lesions was used as inoculum in this group. The tissue was a yellowish-gray and lymphoid-like in

appearance. Histologically the cells were variable in size and shape and were in the undifferentiated blast stage. The bone-marrow was quite severely affected and the blood-picture as observed over a period of days was definitely that of erythroleukosis.

The group contained 38 susceptible Leghorns which were placed in a house as soon as taken from the incubator. Nineteen of these birds were inoculated intraperitoneally with 2 cc of the inoculum when they were ten days old. The remaining 19 birds were left uninoculated purely for pen exposure contact.

In data collected from experiment I, over a period of 13 months, it was found that 60 per cent of the total 38 birds became affected. In the injected group, 14 of the 19 birds were affected (70+ per cent) and in the pen exposure contact group, nine of the 19 birds (50— per cent) were affected.

The positive cases recorded include practically all the previously mentioned manifestations, namely: nerve type, 5 birds; eye type, 1 bird; erythroleukosis, 4 birds; myeloid leukosis, 3 birds; 1 case of mixed eye and nerve type; 4 cases of hemocytoblastosis and 5 cases of lymphocytoma.

This experiment indicates that a transmissible agent was present in the infiltrated organs used as inoculum and that manifestations of erythroleukosis, myeloid leukosis, hemocytoblastic and lymphocytic tumors and infiltrations resulted from a common etiological agent.

The disease was transmitted by intraperitoneal injection of affected tissue suspensions into young chicks and to a less extent by pen exposure contact. In general, the time element between the injected and contact group was different, the contact group taking a longer time. The earliest case to become affected was in the case of a bird developing erythroleukosis in 21 days.

Fifteen birds of the same age and pedigree were used as controls. One of these developed erythroleukosis when about nine weeks of age and one myeloid leukosis when about one year of age.

Experiment II

Transmission by Injection and Pen Contact

A saline suspension made from a markedly enlarged and infiltrated ischiatic nerve was used as injection material in group 2.

The cockerel from which this tissue was obtained showed a complete paralysis of both legs, which had been present for some time. He was extremely weak and emaciated. Blood-smears over a period of two weeks were normal and upon autopsy no gross

lesions, except in the ischiatic nerves, were noted. The nerves were enlarged, edematous, soft and yellowish gray in appearance.

This group of birds consisted of 30 Barred Rocks from our own breeding flock. Fifteen were injected intraperitoneally with 2 cc of the inoculum when one week of age. The remaining 15 birds were left uninoculated for pen exposure contact.

Data collected over a period of 13 months on this group gave us the following results. Eighteen birds of the 30 were classified as positive cases (60 per cent). In the injected group, 11 of the 15 birds were affected (75 per cent), and in the contact group, 7 of the 15 birds or (47 per cent). The positive cases included: erythroleukosis, 3 birds; myeloid leukosis, 1 bird; hemocytoblastosis, 3 birds; nerve type, 5 birds; eye type, 3 birds; lymphocytoma, 2 birds and mixed type, 1 bird.

This experiment, as did experiment I, indicates that the transmitting agent was present in the infiltrated nerve used for injection and that erythroleukosis, myeloid leukosis, lymphocytoma, and nerve and eye types resulted from a single injection of infiltrated nerves.

The 15 controls of this experiment developed one case of myeloid leukosis and one case of eye type. These two cases, however, did not develop until after 13 months.

EXPERIMENT III

Transmission by Injection and Pen Contact

A tissue suspension in saline solution was made from a typical ovarian tumor. The pathological tissue was gray, soft and lymphoid-like in character. Histologically the cells composing the tumorous mass were hemocytoblasts and lympocytes. Blood smears over a period of two weeks were normal and no other lesion, other than that mentioned, was found upon postmortem examination.

Fifteen susceptible Leghorn chicks were inoculated intraperitoneally with 2 cc of this material when they were ten days old. Fifteen chicks were left uninoculated for pen exposure contact. The data obtained from this experiment during a 13-month period revealed the following: 17 of the entire group of 30 birds (61 per cent) were affected, of which eleven of the 15 inoculated birds (78 per cent) and six of the 15 contact birds (40 per cent) were affected.

The following manifestations were recorded: lymphocytomatosis, 7 birds; hemocytoblastosis, 2 birds; myeloid leukosis, 1 bird; erythroleukosis, 3 birds; nerve type, 2 birds; eye type, 1 bird and mixed type, 2 birds. It is to be noted from these experi-

mental data that all the various manifestations resulted from inoculations of the so-called tumor type or lymphocytoma, and that these various manifestations were apparently produced by a common etiological agent present in the tissue suspension of a lymphocytoma.

The control group of this experiment, 15 in number, remained healthy and no cases developed over the 13-month period.

EXPERIMENT IV

Transmission by Injection and Pen Contact by Berkefeld Filtrates

A saline suspension of a large tumorous mass located in the kidney of a bird S was passed through a Berkefeld 3N filter. The tissue was histologically that of a hemocytoblastoma and no other gross lesions were found in the bird. Two cc of this filtrate was injected intraperitoneally into 21 susceptible Leghorn chicks when ten days old. Eight birds were left in this group purely for pen exposure contact.

The data on this group for a 13-month period showed that seven of the 29 birds became affected, or slightly more than 20 per cent, and of this number, six were of the injected group and only one was from the contact group. The following manifestations were noted: erythroleukosis, 2 birds; lymphomatosis infiltration or tumors, 2 birds; nerve type, 1 bird; hemocytoblastosis, 1 bird and eye type, 1 bird.

The percentage of affected birds was much less when filtrates were used than when a tissue suspension was used. It would appear, however, from the number affected that the etiological agent is a filtrable virus and may be obtained from filtrates of affected tissue. In this experiment, as in the others, many of the various manifestations already described occurred from a single inoculum.

The controls of this group were retained 18 months and none of them showed any manifestations of the disease.

EXPERIMENT V

Transmission by Seitz Filtrates

The group of birds in this experiment consisted of 27 susceptible Leghorn chicks, of which 21 were inoculated intraperitoneally with 2 cc of the tumorous material as used in experiment IV except that this material had been passed through Seitz filters instead of a 3N Berkefeld. Six chicks were left uninoculated purely for pen exposure contact.

During the 13-month period, seven of the 27 birds were affected (25 per cent) and all seven were from the injected group. The various manifestations were classified as erythroleukosis, 1 bird; myeloid leukosis, 2 birds; nerve type, 1 bird; lymphocytoma, 3 birds.

The results of this experiment show that there is very little difference in the use of Seitz or Berkefeld 3N filters as far as the percentage of affected birds is concerned. The controls of this group, ten in number, developed two cases of eye type at about eleven months of age.

EXPERIMENT VI

Transmission by Litter Exposure Contact

The house used for this group had been used previously for transmission groups and had housed numerous birds showing the various manifestations of fowl leukosis. The litter, which was ground cobs, and the droppings were not removed from this house during the previous experiments and were allowed to accumulate for two years. The litter was about six inches deep in the house and the only preparation of this house was to rake the litter and dry it.

Thirty chicks of a susceptible strain were placed in this house when taken from the incubator on April 30, 1934.

The tabulations were made over a period of 19 months and birds that were not autopsied appeared in perfect health. Differential smears as well as blood-counts were performed on all birds classified as negative before they were allowed to be put in this classification. It is to be noted that 15 birds, or 50 per cent of the entire group, became affected during the 19-month period. It is also interesting to note that as in the other experiments practically all the various forms or manifestations occurred. The following different expressions occurred: lymphomatous tumors and infiltrations, 3 birds; myeloid leukosis, 2 birds; erythroleukosis, 2 birds; nerve type, 6 birds; eye type, 1 bird and 1 bird with a combination of eye type and hemocytoblastosis.

This experiment indicates that the infection may be quite easily transmitted by contact with contaminated litter or surroundings. This is important to know in instituting any valuable control measures. It also indicates infection may remain in houses or litter for at least two years and probably for a much longer time.

The controls of this group were kept for 19 months and, of the group of 15 birds, one bird developed a lymphoid-like tumorous

mass in the ovary and one a complete paralysis case with infiltration of the ischiatic nerves.

Experiment VII

Transmission Through the Egg

It seems of prime importance and interest to determine whether chicks hatched from eggs laid by affected birds would inherit the disease. The fact that lesions are sometimes found in the ovary, oviduct and testis, suggests that the disease may be spread to their progeny before the function of the reproductory organs is completely checked.

There are many difficulties in securing eggs from paralyzed hens as paralysis causes a cessation of egg-laying in most cases. The selection of birds suffering with internal lesions is a matter of guess as far as clinical diagnosis is concerned. In cases where a definite diagnosis can be made from examination of the blood, other difficulties arise, as most cases when the blood is involved fail to reproduce. In cases where one secures a goodly number of eggs, it is found that most fertile eggs die during the period of incubation.

In this experiment we were able to secure 18 chicks, of which four developed various manifestations of the disease over a period of 18 months.

This experiment was carried on with such a small group of birds that no definite information was obtained.

Summary and Conclusions

1. Fowl leukosis is used to include in common the group of leukemic, aleukemic and leukemic-like diseases of fowls.

2. Young chicks are more susceptible than older ones. The greatest incidence is between the ages of two and twelve months, the majority of cases occurring between four and ten months.

3. The disease in all of its expressions may be transmitted to healthy chicks by injection of tissue suspensions of affected tissues or by injection of cell-free filtrates. It may be transmitted by direct pen contact or by rearing chicks on contaminated litter.

4. Neurolymphomatosis gallinarum, associated with eye lesions, hemocytoblastosis, lymphoid, erythroid and myeloid types of leukosis are different expressions of the same disease and were transmitted by a common etiological agent.

5. Erythroleukosis and myeloid leukosis manifestations are largely dependent upon involvement of the bone-marrow.

6. The lesions may be localized in one area or may be generalized.

7. The injection of a suspension made from one type apparently produces practically all the various types considered as expresssions of this disease. Different suspensions were used in all groups of the experiment but practically the same results were obtained in each group. The incidence is often very high in spontaneous infected flocks.

8. The lesions may be confined to one or more types of tissue in some cases and to some other type or types in other cases. Breed and sex differences apparently do not affect the incidence of the disease.

9. There appears to be an inherent difference of susceptibility and resistance by different birds and birds of different strains.

10. The disease spreads rather slowly in infected flocks and only a few birds are clinically affected at one time.

11. There seems to be some evidence to suggest that the disease may be transmitted through the egg to a limited extent.

12. The evidence indicates that fowl leukosis is an infectious disease and the transmissible agent is a filtrable virus.

13. The clinical course is variable, extending over a period of weeks or months in some cases, while in others it is comparatively short. No complete recoveries occur although temporary respites from the disease have been noted.

14. This disease is one of the most important diseases confronting the poultry industry. The most important methods of control are proper sanitation and the use of breeding stock from resistant sources.

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Doctor Connaway Honored

Nearly 200 persons, representing all parts of Missouri and neighboring states, met at Columbia, on June 22, to honor Dr. J. W. Connaway for his outstanding service in the control of animal diseases. Dr. Connaway's contributions to knowledge in this field were compared to those of Pasteur and other great benefactors of mankind.

Dr. Connaway, in his 49 years of service as a member of the faculty of the University of Missouri, has distinguished himself, said the speakers on this occasion, not only as a scientist but also as a teacher, philosopher, leader and Christian gentleman. Among the speakers were W. A. Cochel, editor of the *Weekly Kansas City Star;* Dr. Frank Nifong, Columbia surgeon; President F. A. Middlebush, of the University of Missouri; and Prof. F. B. Mumford, dean of the Missouri College of Agriculture.

Dr. Connaway has been associated with the University of Missouri as teacher of bacteriology, anatomy and physiology at the Medical School and as Professor of Veterinary Science for 49 years. In point of service to the University this is perhaps the longest period of professional service in the institution. He was first a member of the faculty of the School of Education, then of the faculty of Arts and Science and, since 1891, has been a member of the faculty of Agriculture, Veterinary Division.

The speakers mentioned Dr. Connaway's achievements in veterinary science, particularly the work done on Texas fever, hog cholera, and Bang's abortion diseaee.

His friends and colleagues of the College of Agriculture presented him with a scroll, containing the names of all those at the banquet, with a suitable inscription. In addition a beautiful plague was presented, which is to hang in the hall of the Veterinary Building with the following inscription:

IN HONOR OF
JOHN WALDO CONNAWAY, D. V. S., M. D.
PROFESSOR OF VETERINARY SCIENCE AND COMPARATIVE MEDICINE
UNIVERSITY OF MISSOURI
IN RECOGNITION OF HIS DISTINGUISHED SERVICE
AS TEACHER, SCIENTIST AND CITIZEN
BY FRIENDS AND COLLEAGUES
JUNE 22, 1937

STUDIES ON BOVINE BLOOD

I. The Sedimentation Rate and Percentage Volume of Erythrocytes in Normal Blood*†

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Department of Veterinary Science
University of Wisconsin

A survey of the literature on hematology has failed to reveal any serious attempt to establish the normal variation in the suspension stability of erythrocytes in the blood of normal cattle. Likewise, the percentage volume of erythrocytes in bovine blood has not been investigated to any great extent. Dukes,[1] in 1935, quoting Bürker, reported a normal value of 32 per cent, but at the same time emphasized the need of securing more knowledge relative to the individual hematological variations in normal cattle.

The purpose of this paper is to present the results obtained from a study of the sedimentation rate and percentage volume of erythrocytes in a herd of disease-free cattle in an effort to determine the normal range of variation within and between the individual animals. This study has been carried on in connection with a Bang's disease project, in which the normal hematology is being determined for a basis of comparison to the hematological variations which may result when these animals are infected with *Brucella abortus*.

THE SEDIMENTATION RATE

The suspension stability of erythrocytes in blood, to which an anticoagulant has been added, has been the basis of much clinical and investigational work since Fahraeus,[2] in 1918, presented the test as a means of diagnosing pregnancy in women. Since that time, hundreds of papers have been published on technics, applications and interpretations of the test. The sedimentation test is now recognized as an important clinical aid in following the course of certain pathological conditions of man, especially tuberculosis.

The exact cause for the variation in the suspension stability of the erythrocytes in the various species, as well as in pathological

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936. Published with the permission of the Director of the Wisconsin Agricultural Experiment Station.

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conditions of the same individual, is not definitely known. The results secured by many workers indicate that the sedimentation rate increases proportionately with the amount of tissue destruction. In acute infectious diseases, carcinoma, severe local infections, tuberculosis, and other diseases, the rate of sedimentation closely parallels the amount of organic tissue destruction. Fischel[3] reports that in unilateral pulmonary tuberculosis successful compression of the affected lung by artificial pneumothorax results in an immediate decrease in the sedimentation rate. This is apparently due to the decrease in tissue destruction following the inactivity of the diseased part.

Greishheimer, Van Winkle and Johnson[4] have shown that there is a significant negative correlation between the sedimentation rate and the fibrin content of the same sample of blood. The number of erythrocytes per cubic millimeter of blood has a definite effect on the rate of sedimentation. Boerner and Flippin[5] corrected the red cell count to about normal in 50 blood samples which showed rapid sedimentations and of these 14 were normal after the anemic condition of the blood was corrected. Rourke and Ernstene[6] showed that the sedimentation rate is inversely proportional to the percentage volume of erythrocytes, and they constructed a chart for clinical use to correct for the anemic condition of the blood. For further discussion and references to factors affecting the sedimentation rate, see Rourke and Plass,[7] Cherry,[8] Wintrobe and Landsberg,[9] and Cutler.[10]

Many technics have been described for determining the rate of sedimentation of the erythrocytes. The technic first described by Fahraeus[2] and modified by Westergren[11] has been used extensively. Cutler[10] developed the "graphic" method for the determination of the sedimentation rate of human blood. The technic consists of using 0.1 cc of three per cent sodium citrate to 0.9 cc of blood. This citrated blood is placed in the Cutler tube, which is graduated in millimeters from 0 at the 1-cc mark to 50 at the bottom of the tube. Readings are made at five- or ten-minute intervals for one to two hours. The results may be recorded on a special form in such a way as to make direct comparison of the sedimentation curve to the established normal and pathological rates.

Percentage Volume of Erythrocytes

The relation of the volume of erythrocytes to the entire column of centrifuged blood, stated in percentage, is called the percentage volume of red blood cells. This value follows rather closely the total red cell count of a particular individual so that one might

expect that the usual factors which affect the red cell number would also change the percentage volume.

In the percentage volume determinations by the hematocrit method the speed and length of centrifugation is a very important factor. Heller and Paul[12] found that cattle blood cells reach a contant volume after centrifugation for 45 minutes at 2,200 r.p.m. Turner and Herman[13] used 45 minutes at 2,800 r.p.m. The inorganic salts, commonly used as anticoagulants, have a marked but constant effect on the cell volume. Heller and Paul[12] have shown that potassium, sodium, and lithium salts of oxalic and citric acids decrease the cell volume as the concentration increases while ammonium salts tend to increase the cell volume.

Wintrobe[14] described a simple, accurate hematocrit method for cell volume determination. He used an 11-centimeter, flat-bottomed, graduated tube with a 3-millimeter bore. Ten mg of dry potassium oxalate were used as the anticoagulant for 5 cc of blood. The samples were centrifuged until a constant cell volume was reached. Wintrobe and Miller[15] found that the addition of 20 mg of potassium oxalate to 10 cc of heparinized blood caused 3.68 per cent shrinkage of the cells as compared to the heparinized blood. The final results were therefore increased by 3.68 per cent.

Experimental Procedure

The animals used in this experiment were all normal Holstein-Friesian cattle varying in age from three to eight years. This herd has been tested at regular intervals for both tuberculosis and Bang's disease and found to be free of these infections. The cows were in various stages of pregnancy and lactation. Observations on animals showing definite clinical symptoms of disease of any kind have been excluded. The blood samples for routine testing were drawn from the jugular vein into tubes of 25-cc capacity containing 0.1 cc of 20 per cent sodium citrate solution. The minimum amount of citrate necessary to prevent coagulation was found to be 10 mg for 5 cc of blood. Since the amount of blood drawn varied from 6 to 9 cc, 20 mg of citrate were used. The blood was well mixed by closing the blood-tube with a paraffin-coated cork stopper and tilting gently from end to end. By means of a capillary pipette the citrated blood was transferred to a Cutler tube,[10] filling it to the 1-cc mark. The blood was covered with a layer of liquid paraffin to prevent evaporation and to facilitate reading. Observations were made at hourly intervals since it was found that more frequent readings did not increase the accuracy of the results. Readings extended over an eight-hour period. At the end of this time, the tubes were centrifuged

in 15-cc cups (International centrifuge, size 1, head 225) for 25 minutes at 3,200 r.p.m. It was found that four of the Cutler tubes fit into each centrifuge cup in such a way that no special attachment is needed. This procedure packs the cells to constant volume, as is illustrated in table I. The height of the cells and of the entire blood column is noted and the results recorded.

TABLE I—*The effect of different periods of centrifugation on the volume of packed cells.**

Cow	Centrifuged at 3,200 r.p.m. for					
	10 Min.	15 Min.	20 Min.	25 Min.	30 Min.	35 Min.
1	19	17.0	15.0	13.5	13.5	13.5
2	22	18.5	17.0	16.0	15.7	15.7
18		17.5	16.0	15.5	15.5	15.5
20		13.0	12.0	12.0	12.0	12.0
24		15.0	14.5	14.0	13.9	13.9
25		15.0	14.5	14.0	14.0	14.0
27		13.5	13.0	13.0	13.0	13.0
28		16.0	15.0	14.5	14.5	14.5
29		14.0	13.0	13.0	13.0	13.0
30		15.0	14.0	13.5	13.3	13.3
32		14.5	13.5	13.5	13.5	13.5
58		15.0	14.0	14.0	13.9	13.9
18				17.0		17.0

*The values in the table are given in millimeters.

In order to calculate a correction factor for the shrinkage of the erythrocytes, the technic of Wintrobe and Miller[15] was followed. From a series of tests it was found that the citrate caused 6.5 per cent shrinkage in volume of the red cells. This value was used in calculating the corrected percentage volume which has been recorded in all of the tables.*

RESULTS

Observations have been made on the blood of 22 cows over a period of three months to determine the sedimentation rate and the percentage volume of erythrocytes. The sedimentation rate of 423 blood samples has been included. For convenience in presenting the results the *sedimentation index* has been defined as the number of millimeters which the erythrocytes have settled at the end of seven hours. As shown by the curves in figure 1, the sedimentation index represents a value which is characteristic of the sedimentation rate.

*The methods used in this calculation are given in detail in the author's thesis submitted for the degree of Master of Science, University of Wisconsin, 1936.

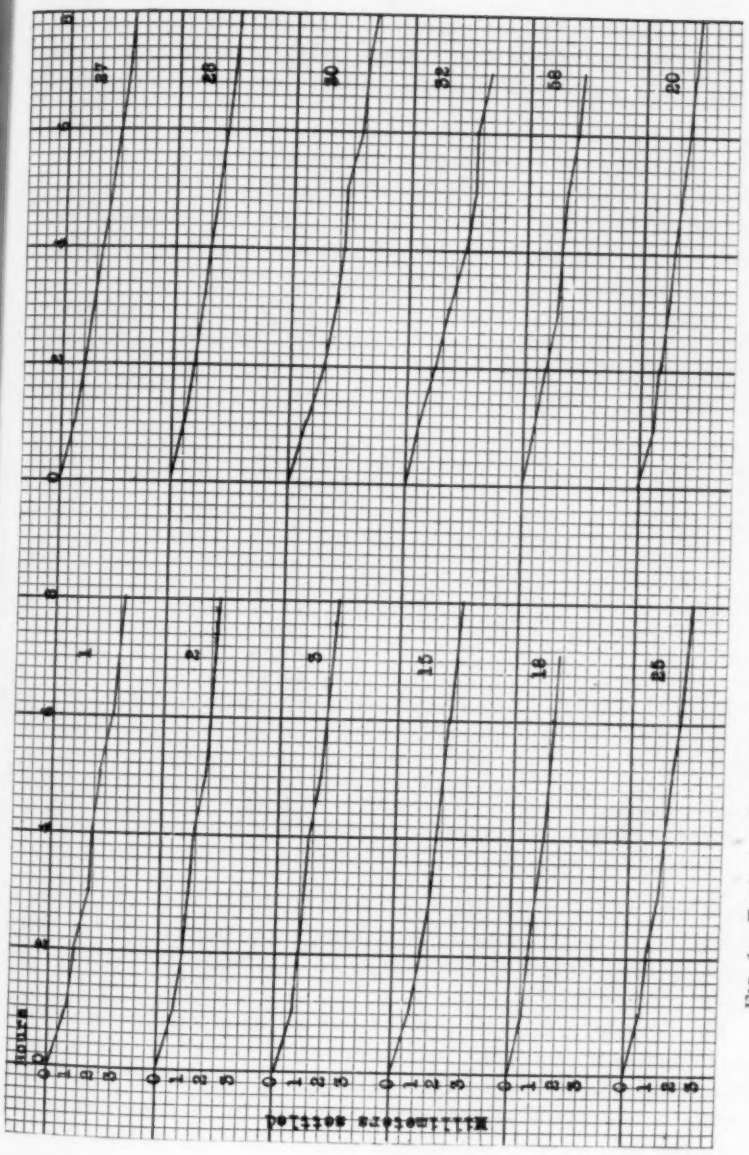

FIG. 1. Typical sedimentation curves of the blood from the normal cows examined.

In order to present the general type of the sedimentation curve for cows, a number of individual observations are presented in figure 1. These have been chosen at random from the data so that the extreme as well as the average rates of settling are represented. The mean sedimentation index has been calculated for

TABLE II—*The mean percentage volume of erythrocytes and the mean sedimentation index of each cow.*

Cow	Percentage Volume	Sedimentation Index (Millimeters)
1	29.89	2.52
2	34.53	2.34
3	31.48	2.17
6	33.46	2.17
7	34.11	2.09
8	31.37	2.25
11	29.19	2.22
12	31.74	2.30
13	30.00	2.44
15	32.47	2.26
63	31.64	2.07
18	37.83	1.72
20	31.03	2.39
24	28.71	2.60
25	30.44	2.48
27	29.10	2.62
28	29.89	2.76
29	32.40	2.50
30	28.95	2.64
31	31.88	2.50
32	29.60	2.99
58	29.28	2.64
Mean of the means	31.32	2.394
Standard deviation	2.194	0.275

each cow and the results are listed in table II. The mean of these individual means, 2.394 millimeters, represents the mean sedimentation index of the herd. The standard deviation of the mean value is 0.2750.

This standard deviation, as well as a casual inspection of table II, indicates a relatively large amount of variation. In order to determine the source of this variation the data have been subjected to a statistical analysis using the method of analysis of variance as described by Snedecor.[16] This is a technic for segregating from comparable groups of data the variation arising from specified sources. The calculations consist of determining total variance of the population, the variance between individuals and the remaining variance, which includes that within individ-

uals as well as the experimental error. The word "variance" denotes the square of the standard deviation. The term, "mean square" is often used instead of "variance."

The major portion of the calculations in analysis of variance is designed to give the sum of the squares of the deviations of the observed values from their mean. This quantity is designated in table III by the abbreviated phrase, "sum of squares."

The degrees of freedom, as explained by Fisher,[17] represent the number of individuals or classes decreased by one. This value is used instead of the true number because, in calculating the mean, one degree of freedom is sacrificed. For example, in table III the total number of observations is 423 and the number of degrees of freedom is 422. Likewise the number of individuals, 22, less 1, leaves 21 degrees of freedom. The number of degrees of freedom within individuals may be taken as the difference between the "total" and the "between individuals," $422 - 21 = 401$.

TABLE III—*Analysis of variance of the sedimentation index.*

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARES
Total	422	79.21	0.188
Between individuals	21	28.41	1.353
Within individuals	401	50.79	0.127

The mean squares were calculated by dividing the "sum of squares" by the corresponding number of degrees of freedom. A summary of analysis of variance of the sedimentation index is given in table III.

Examination of the summary shows that the total variance has been divided into two parts; first, that due to the differences between individuals, and, second, that due to the variation within individuals. As explained above, the *mean square* within individuals represents the average variance of the population and may be used for significance tests. The variation between individuals is compared to that within individuals by use of the *mean squares.*

$$F = \frac{\text{larger mean square}}{\text{smaller mean square}}$$

$$\text{Therefore} \quad F = \frac{1.353}{0.127} = 10.6$$

Using Fisher's table of F (Fisher[17]), with the degrees of freedom indicated in table III, the 1 per cent tabular value of F is 1.84. Since this is exceeded by the calculated value of F, 10.6, the differences between individuals in this experiment are considered highly significant, that is, they would occur due to chance alone in less than 1 per cent of the cases if the same experiment were repeated.

In order to show the marked effect of certain pathological conditions on the sedimentation rate of bovine blood, a case report is included. Cow 25 developed an acute, local infection in the subcutaneous tissue around the tarsal joint. A definite increase in the sedimentation rate was noticed before the herdsman recognized the affection. The rate of sedimentation is indicated by the curves in figure 2. At the height of the disease, the sedimentation index was 17.5 mm, as compared to her normal, mean index, 2.48 mm. When the severity of the infection had decreased, as indicated by clinical symptoms, the sedimentation rate returned to normal, the entire course of the affection lasting less than two weeks.

The percentage volume of erythrocytes in 439 blood samples from 22 cows has been determined. The mean percentage volume of cells for each cow is listed in table I. The mean of these individual means, 31.32 per cent with its standard deviation, 2.194 per cent, represents the mean percentage volume of the population.

In order to determine the source of the variation in the percentage volume determinations, the data were subjected to analysis of variance as described in connection with the sedimentation index. A summary of the results obtained is given in table IV.

Examination of table IV shows that the total *sum of squares* has been divided into two parts, that due to individual variation and the remainder which represents the average variability around the individual mean values. This latter part of the variance is attributed to the normal or "physiological variation," as well as the experimental error. To compare the variation between and within individuals, the mean squares are used as before.

$$F = \frac{35.449}{2.185} = 16.2$$

Here, again, this value of F greatly exceeds the 1 per cent value in Fisher's table, thereby indicating a highly significant differ-

TABLE IV—*Analysis of variance of percentage volume of erythrocytes.*

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARES
Total	438	1,655.54	3.780
Between individuals	21	744.43	35.449
Within individuals	417	911.11	2.185

ence between individuals. In other words, the variation between individuals is much greater than that which occurs due to variation in the individual from day to day or to experimental error.

Cutler,[10] Boerner and Flippin,[5] Rourke and Plass,[7] as well as other investigators cited by these workers, have reported a correlation between the percentage volume, or the red cell count, and the sedimentation rate. In order to determine if this relation exists in the blood of cattle, the correlation coefficient for each individual was calculated according to the method outlined by Wallace and Snedecor.[18] The values ranged from a positive correlation of 0.250 to a negative correlation of 0.716. With the exception of the one cow (2) which showed the positive correlation, all of the individuals gave a negative value. Although the values taken individually are not statistically significant, there is a definite indication of a tendency toward negative correlation.

DISCUSSION AND SUMMARY

The sedimentation rate of the blood of cows is very slow as compared to man and certain other mammals. The mean values obtained for the sedimentation index of the 22 cows examined varied from 1.72 to 2.99 mm, with a mean value for the group of 2.394, with a standard deviation of 0.275. The lower extreme is represented by cow 18, which has consistently shown a very slow rate of settling. The highest extreme was shown by cow 32, with a value of 2.99 mm. She has shown a low red cell count and a low value for percentage volume of erythrocytes (1.72 per cent below the mean). Exclusion of the two cows showing the extreme variation leaves the range from 2.07 to 2.76 mm, which is less than three times the standard deviation.

The statistical analysis has indicated a rather significant difference between the individual cows as compared to the variance resulting from individual variation and experimental error. This analysis indicates that even though the value for the sedimentation index is relatively quite low in cows as compared to man, it is nevertheless an accurate measurement of individuality.

The mean value, 2.394 mm, or the range, 1.72 to 2.99, represents the approximate normal limits of the sedimentation index for the herd of cattle examined. Since these cows are comparable to the average dairy unit, it is probable that this value for the sedimentation index may be considered as normal for the blood of cows.

The possible rôle of the fibrin content of the blood serum as a factor in determining the rate of sedimentation has been considered. The fibrin content of the plasma of cows is relatively much higher than the normal value for man. Howell[19] gave the normal value of fibrinogen in man as 0.42 gm in 100 cc of serum. Howe[20] reported that the serum of cows contains about 0.72 gm of fibrinogen per 100 cc. Greishheimer et al.[4] have shown a significant negative correlation between the sedimentation rate and the fibrin content in man. Comparing the relatively slow sedimentation rate of the cow to the more rapid rate in man, it seems reasonable to conclude that the fibrin content of the serum may be of importance in determining the rate of erythrocyte sedimentation. The correlation may, however, be only accidental, so we should withhold final conclusions until more definite and critical experiments are completed.

The highly significant difference obtained between individuals in the analysis of variance of percentage volume indicates a variation similar to that in the sedimentation index. The mean of the entire herd, calculated from the individual means, for percentage volume of erythrocytes, is 31.32 per cent with a standard deviation of 2.194. The mean values range from 28.71 to 37.83 per cent. As with the sedimentation index, it is probable that this value represents the normal for dairy cattle. Three times the standard deviation on either side of the mean will include over 99 per cent of the means of any group of cell volume determination made in this same manner. Therefore, a mean derived from a similar group of data on the blood of normal cattle will fall between 24.738 and 37.802 per cent (31.32 ± 6.582).

The correlation coefficients calculated for the sedimentation index and the percentage volume of erythrocytes show considerable variation, but there is some indication of a negative correlation when the group is considered as a whole.

The marked increase which may occur in the sedimentation rate of the red cells in the blood of cattle affected with certain pathological conditions, as illustrated in figure 2, indicates that it may be of practical value as a further aid in the diagnosis and prognosis of certain infectious diseases of cattle. A percentage

volume determination on the same sample of blood also gives an indication as to the relative volume of erythrocytes, which will be of especial value if a total red cell count is not made. It is believed that the combination of these two tests, when applied to the blood of cattle, will be helpful in determining the amount of tissue destruction in local or generalized disease. A sedimentation index of over 4 mm is considered as indicative of a pathological condition.

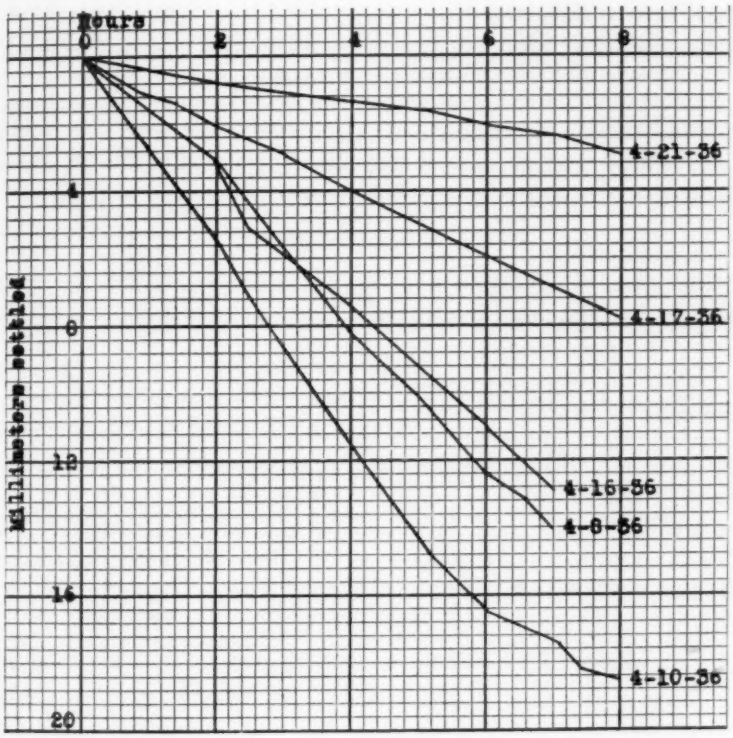

FIG. 2. The sedimentation curves of the blood of cow 25 during the course of an acute infection.

CONCLUSIONS

Studies on bovine blood, including the sedimentation rate and the percentage volume of erythrocytes, have been made on 22 cows. The mean sedimentation index of the herd of cattle, calculated from the individual means, is 2.394 mm, with a standard deviation of 0.275 mm. A value of over 4 mm is considered as indicative of pathology.

The mean percentage volume of erythrocytes, calculated from the individual means, is 31.32 per cent, with a standard deviation of 2.194 per cent.

The results of analysis of variance with both the sedimentation index and the percentage volume indicate a highly significant difference between individuals as compared to that within individuals.

The correlation coefficients of the sedimentation index and the percentage volume of erythrocytes possibly indicate a significant negative correlation.

The relatively high fibrin content of normal bovine serum may account for the slow sedimentation rate.

Since the sedimentation rate is increased in certain pathological conditions, application of the sedimentation test and percentage volume determinations may be of practical value as an aid in diagnosis and prognosis of certain cattle diseases.

Acknowledgment

The author wishes to express his appreciation to Dr. F. B. Hadley and Dr. M. R. Irwin for their helpful criticism of this work.

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DISCUSSION

DR. H. J. METZGER: In your work, did you use any samples of blood from cows immediately before or immediately after calving?

DR. FERGUSON: Yes. We had a few samples of that type. There is a slight increase in the rate just before calving. However, if the calving is normal, usually the rate comes right back to normal, and there is never any marked disturbance. There is usually some slight increase, which may be noticeable a week or even longer before calving.

DR. A. F. SCHALK: Have you conducted any experiments on or noticed any change in relation to cases of mastitis?

DR. FERGUSON: Yes, we have. We have used some tests in that connection on cases of mastitis. We have found in chronic cases that there may be a rate which is slightly above normal.

We have taken as a pathological indication a sedimentation index of 4 mm, and we have had some chronic cases which give a rate that high. We also checked cases of acute mastitis and there we find quite a marked increase, the sedimentation index rising to 5, 6, and 7 mm.

Area Testing for Bang's Disease in Wisconsin

The signing of Wisconsin's Bang's disease area test bill by Governor Philip F. LaFollette, at the close of the 1937 session of the legislature, was witnessed by the author of the bill and officials of the State Department of Agriculture and Markets who will administer its provisions: Assemblyman William H. Barnes, of New Lisbon, who introduced the bill and steered it to final passage; Commissioners Charles L. Hill and F. Schultheiss, of the Department of Agriculture and Markets; and Dr. W. Wisnicky, Director of Live Stock Sanitation, who watched Gov. LaFollette affix his signature to this major piece of farm legislation. The bill which was passed by a vote of 73 to 15 in the Assembly and 27 to 2 in the Senate, provides for area testing, which would involve all cattle in a county after 75 per cent or more of the herd-owners in such county have petitioned the Department of Agriculture and Markets for such a test. Cattle condemned under the area test procedure will be disposed of and the herd-owner will receive both state and federal indemnity in addition to net meat salvage. Several counties are now having petitions signed in order that they may take advantage of the provisions of the new law.

A woman likes to spend a long time over her hair and a man likes to spend a long time under his.

RENGUERA*

By J. F. MITCHELL, *Pachacayo, Peru*

Synonyms: Paraplegia enzootica (Span.), paraplegia enzootique des agueaux (Fr.), louping ill and swingback.

Definition: Renguera is a paraplegia of lambs, kids, llamas, alpacas and vicunas, caused by a filtrable, infectious and inoculable virus which attacks the central nervous system, causing locomotor ataxia.

HISTORY: Renguera was studied first in 1911, by Dr. L. Maccagno. This outbreak occurred around Cerro de Pasco, in central Peru. There are records of earlier outbreaks, but they were not studied by professional men. In the 1911 outbreak about 90 per cent of the lambs died. A second outbreak occurred from 1915 to 1918; a third from 1923 to 1926; a fourth, of much less importance, in 1930; a fifth outbreak is now in progress (December, 1936). The present outbreak is quite severe, as about 30 per cent of the lambs are being attacked. A large number of the ewes have aborted and the losses are running up into the thousands of lambs.

Dr. M. E. Tabusso began studying renguera in 1911 and continued until 1920. During that time he published two bulletins[1, 2] on the history, symptoms and pathology of renguera. He did not discover the cause of the disease. Dr. S. A. Gaiger studied renguera in 1916. He isolated a diplococcus from which a vaccine was made. However, the vaccine did not prevent renguera. Later, Dr. Gaiger made the statement that renguera in Peru and swingback in England are not clinically distinguishable from one another.

Dr. Fritz Mrowka studied renguera in sheep and cattle in northern and central Peru, but came to no definite conclusion in regard to renguera in sheep. Renguera in Peru kills thousands of lambs during the epizoötic years. Between such epizoötics the disease dies down and there are only a few sporadic cases now and then.

Geographical distribution: Renguera occurs in the Andean regions of central and northern Peru. It has been reported in southern Peru. The elevation of these regions above sea level varies from 10,000 to 19,000 feet. The nights are cold and the days are hot. The annual rainfall is around 20 inches.

Animals affected: Lambs and kids, from two weeks to four months old, are most commonly affected. Older sheep and goats

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seem to be immune. Younger lambs are sometimes attacked and a few are born with the disease. Abortions in the ewes may occur during the epizoötics. Young alpaca, llamas and vicunas are often affected. The people in the region handle the cases with impunity. The flesh is eaten and it seems to do no harm to those consuming it. Cases of infantile paralysis occur in the children in this region, but the disease is not particularly common.

FIG. 1. A case of renguera due to natural infection. Note the position of the hind legs. The fore legs are in normal condition in this case but they may be attacked.

ETIOLOGY

I have proved that renguera is caused by a filtrable virus by reproducing the disease in 138 animals. I have passed the disease through three generations. For virus I have used the ground brains and spinal cords of lambs and kids affected with renguera. About 65 per cent of the lambs injected with

this material, after it has been filtered, develop the paralytic walk of "renguered" animals. Injections have been successful in transmitting the disease, when made into the brain, nose, spinal cord and peritoneal cavity. Injections of blood or of extracts of any other organs have failed to transmit renguera. Subcutaneous, intramuscular and oral administrations of virus have failed to transmit the disease.

Methods of Infection

The natural method of infection with renguera is not known. Paralytic cases of renguera may live for years with supposedly susceptible lambs and not transmit the disease. In epizoötic years, nearly all of the lambs contract renguera. I have found sheep ticks on a great many natural cases of renguera, but on lots of other cases there were no ticks at all. Lambs less than a month old rarely have ticks, but quite often they have renguera.

Blood from the ordinary paralytic case is not infectious, even in 100-cc doses. However, it may be infectious during fever. Quite often there are two stages of fever after inoculation. The first febrile period comes on ten days to two weeks after injection and passes off without leaving noticeable symptoms. The second fever comes on in one or two months after inoculation and it is following this second febrile period that paralysis develops in about 65 per cent of the cases.

The following animals have been injected without causing renguera: rabbits, guinea pigs, dogs, cats, calves, alpacas, llamas, three monkeys and two deer. So far I have not been able to obtain mice or rats for inoculation. It is peculiar that I have not been able to produce a case of renguera in either alpacas or llamas, although natural cases occur quite often in these animals.

Attempts to cultivate the causative organism on artificial culture media have failed.

Serum or defibrinated blood from recovered or repeatedly inoculated cases of renguera does not have any curative effect on the paralytic cases, nor does it prevent paralysis.

Period of incubation: The shortest period of time between inoculation and the development of paralysis was 21 days, and the longest was four months. The average has been 2½ months. Under natural conditions a few cases are present at birth and a few more develop during the first few weeks of life, but the majority of the cases develop in lambs between the first and fourth months.

Metallic poisoning: Owing to the fact that lead sometimes causes a paralysis in people, there developed a theory that lead

was causing the paralysis in the lambs. I therefore repeatedly tried to cause renguera by feeding lead, arsenic and flue dust to ewes and lambs. In some cases the feeding was spread over a short time; in other cases months, to simulate natural conditions. These experiments resulted in complete failure. I was not able to produce a single case of renguera in this way, even when I fed the poisons to the ewes before lambing and to the lambs after they were born. On the contrary, a few cases recovered when given Fowler's solution of arsenic. Chemical analysis of the milk of ewes whose lambs had the renguera failed to show lead.

FIG. 2. A case of renguera due to the intraperitoneal injection of virus. Note the position of the hind legs and feet. The fore legs are normal.

SYMPTOMS

"Renguera" is taken from the Creole word "rengo," meaning lame or to wobble or sway when one walks. It is very appropriate for this disease. After walking for a short distance, the lambs gradually lose muscular control. They can not pull their legs forward, they sway more and more from side to side, and finally swing over to one side in a sitting posture, or lie prone on the ground. After a rest of a few minutes, a lamb may rise and travel for a short distance.

Some of these lambs are so nearly healthy that, when one sees them standing still or walking slowly, they appear to be normal. The worst cases can not stand up. Many of them can not extend their toes. As a result they walk on their knuckles and drag their toes over the ground. Most of the cases can stand travel and are able to eat. When on good pasture and not far from water, such animals live on indefinitely and a few recover. I have a flock of 62 ewes, all of which were paralytic. Forty-four are still paralytic and 18 have recovered. All have given birth to two or three lambs. Their lambs came normally and may or may not develop renguera later on. The great majority of the lambs affected with renguera are killed by condors or foxes, or die from starvation or by drowning.

The temperature is normal except during the fever curve of the second week and just before paralysis sets in. The sensibility of the skin over the affected limbs may or may not be lost. Except in extreme cases there is no shivering or trembling until the animal is completely tired out. The affected muscles are slightly shrunken. The fore as well as the hind limbs may be attacked.

Morbid anatomy: Macroscopic lesions are largely lacking. The brain and cord may be reddened or they may appear normal. The heart, lungs, liver, kidneys and intestines appear normal. Cultures are negative or may yield a diplococcus. Filtrates of the spinal cord and brain remain clear. Such filtrates will cause paralysis in about 65 per cent of the lambs injected with it.

Treatment

The individual medication of several hundred lambs out on the range is not practical, especially at lambing time. A very small percentage of these paralytic lambs will recover without treatment. A few more will recover if put on very good pasture near water. In corral-fed lambs I have cured several cases with each of the following drugs: Fowler's solution of arsenic; quinine sulfate; iron, quinine and stychnine citrate; methylene blue; neosalvarsan; salvarsan, and red iodide of mercury. I secured the best results with the intravenous injection of mercurochrome, to which digitalin had been added. Sixty-eight per cent of the lambs so treated recovered. The treatment of old chronic cases is very unsatisfactory.

Prophylactic Measures

Serum and defibrinated blood from recovered and repeatedly injected cases have been used both as a curative and as a preventive. The results have been indefinite.

Vaccination to prevent renguera is being tried. The vaccine is made by grinding the spinal cords and brains of affected lambs, adding physiological salt solution, and treating the emulsion with chloroform or formalin, after the method of making rabies vaccine. The results so far are not very definite. It will take at least one more lambing season to be sure of the results. They are encouraging but not definite.

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California to Complete Tuberculosis Eradication

The Legislature of California recently provided a special appropriation of $1,500,000 for tuberculosis eradication to be extended to all counties of the state. The appropriation is for two years and the work is to be conducted coöperatively with the United States Bureau of Animal Industry. An enlarged program will be instituted at once, requiring an additional number of veterinarians. It is hoped that, by the end of the two-year period, eradication will practically be complete in the entire state.

There are 58 counties in California, of which 20 are now modified accredited. The status of the remaining 38 counties varies all the way from some in which an eradication program has not yet started to others nearly eligible for modification.

California was late in beginning eradication work. A decision of the State Supreme Court first was necessary to establish the constitutionality of indemnity payments for reactors. The large cattle population with a comparatively high percentage of infection made the program a big undertaking. Eradication in a number of counties has been impeded by an organized opposition to the work. C. U. D.

Bureau Transfers

DR. WILLIAM C. GLOCKNER (U. P. '20), from Baton Rouge, La., to Frankfort, Ky., on tuberculosis eradication.

DR. HENRY J. GOHDE (K. C. V. C. '17), from Chicago, Ill., to Austin, Minn., on meat inspection.

DR. CLARENCE B. HENDERSON (K. C. V. C. '09), from Leavenworth, Kan., to Topeka, Kan., in charge of meat inspection.

DR. OLIVER W. ORSON (Tex. '32), from Pittsburgh, Pa., to Spokane, Wash., on meat inspection.

DR. E. H. NORDSTRUM (Iowa '29), from Oklahoma City, Okla., to Chicago, Ill., on meat inspection.

STUDIES ON HEMORRHAGIC SEPTICEMIA*

By J. B. TAYLOR, *Brookings, S. Dak.*
South Dakota State College and Experiment Station

GROUPING AND IDENTIFICATION OF CULTURE

The culture used in this study was a buffalo strain marked "Buffalo B," obtained from the Bureau of Animal Industry, Washington, D. C. The results of Gram-staining showed it to be Gram-negative. Tests for bile solubility were negative when 1 cc of ox bile was added to 10 cc of a 24-hour bouillon culture and incubated for two hours. Smears showed the organisms to be unchanged. Bouillon with a reaction of pH 7, containing 1 per cent of the various sugars in fermentation tubes, was inoculated and incubated for seven days. No gas was found in any of the tubes. The following was the change in pH value: Lactose, pH 7; dextrose, pH 6; maltose, pH 7; mannite, pH 6.1; sucrose, pH 6.8; salicin, pH 7. There was no addition of serum to any of the tubes. The above tests show that the buffalo B strain belongs to Jones' group III.[1]

TOXIN PRODUCTION

The production of exotoxin: Buffalo B strain of hemorrhagic septicemia was grown in beef-heart broth pH 7 for seven days, since too long a cultivation might produce endotoxin as well as exotoxin. The bouillon culture was then filtered by passing it through filter paper and sterile Berkefeld filters grade V (coarse) and then grade W (dense). One cc of the filtrate was inoculated into 50 cc of beef-heart broth pH 7 and incubated for 48 hours to determine if the filtrate was sterile. When found to be sterile it was injected intravenously into rabbits weighing about 5 pounds each; in doses of 0.5 to 3.0 cc it caused death and below 0.5 cc all rabbits survived.

The production of endotoxin: The Buffalo B strain of hemorrhagic septicemia was grown on beef-heart agar for 24 hours, washed off with sterile distilled water into sterile flasks and incubated for 24 hours at 37.5° C. The flasks were put in a shaking-machine for one hour and again incubated for another 24 hours. This treatment should bring about autolysis and liberation of endotoxin. The heavy suspension of organisms in distilled water was then passed through filter paper and sterile Berkefeld filters of grades V and W. A number of loopfuls of the filtrate were planted on beef-heart agar to determine if the

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filtrate was sterile. The sterile filtrate was then inoculated intravenously into rabbits ranging around 5 pounds each. In doses of from 1.0 to 3.0 cc the filtrate killed the rabbits; in doses below 1.0 cc the rabbits survived.

The Development of a Toxoid

The Buffalo B strain of hemorrhagic septicemia was grown on beef-heart broth pH 7 for ten days. It was then passed through filter paper and sterile Berkefeld filters V and W grades. The filtrate was then treated with 1 per cent formalin solution and incubated at 36° C. for five days. The toxoid was then precipitated out of the filtrate by adding two volumes of acetone while the filtrate was at 4° C. The precipitate was collected and weighed and then dissolved in 0.85 per cent salt solution at the rate of 2.0 gm of precipitate to 100 cc of salt solution. Rabbits were injected with 2.0 cc of this product intramuscularly and showed no distressing symptoms. Fifteen days later, each rabbit weighing about 5 pounds was injected subcutaneously with 0.1 cc of a 24-hour bouillon culture of Buffalo B strain. All rabbits died.

Biological Products and Immunity

A number of rabbits approximately the same size and full grown were caged in groups and given two doses of 1 cc each, at 5-day intervals, of hemorrhagic septicemia aggressin and whole culture chemically killed bacterin, and the following biological products prepared by the author from two buffalo strains of hemorrhagic septicemia: saline bacterin killed with 1 per cent formalin, whole bouillon bacterin killed with 1 per cent formalin, and whole bouillon bacterin with addition of 1 per cent sodium ricinoleate. Three weeks after the last injection of these products, the rabbits were inoculated with 0.1 cc of a 24-hour culture of *Pasteurella bubaliseptica*. All the rabbits receiving the biological products prepared by the author and elsewhere died.

Tests for Solubility of the Organisms of the Hemorrhagic Septicemia Group

Several strains of each of the following organisms were treated with solutions of bile, saponin, sodium oleate, cholesterol, sodium ricinoleate, and sodium desoxycholate for a two hour period: *Pasteurella oviseptica*, *P. aviseptica*, *P. suiseptica*, *P. boviseptica*, *P. equiseptica* and *P. bubaliseptica*. Microscopic examination was then made from the various cultures tested. None of the Pasteurella strains was soluble in the solutions mentioned above.

Sodium Bicarbonate in Hemorrhagic Septicemia

There have been a number of case reports by veterinarians on the favorable results obtained from the use of sodium bicarbonate in hemorrhagic septicemia. Five rabbits weighing about 5 pounds each were injected with 0.1 cc of a 24-hour bouillon culture of Buffalo B strain of hemorrhagic septicemia. One hour later, the rabbits were given an intravenous injection of 0.5 gm of sodium bicarbonate in 5 cc of sterile distilled water. The same dosage was repeated about six hours later. All rabbits developed hemorrhagic septicemia and died.

Neoarsphenamine in Hemorrhagic Septicemia

Five rabbits were given 0.1 cc of a 24-hour bouillon culture of Buffalo B strain of hemorrhagic septicemia. One hour later, they were given 0.008 gm of neoarsphenamine per kilo by intravenous injection. All rabbits developed hemorrhagic septicemia and died.

Pathogenicity of Strains of Hemorrhagic Septicemia

About twelve isolations of *P. oviseptica* were made from the heart-blood of sheep showing hemorrhages on the heart and pneumonia, mostly of the apical lobes of the lungs. When these strains were cultured and inoculated into rabbits, they were not pathogenic enough to produce death. A number of strains of *P. boviseptica* isolated from blood sent in by practitioners from cattle supposedly suffering from hemorrhagic septicemia were inoculated into rabbits. All but a very few were devoid of pathogenicity. A strain of hemorrhagic septicemia organism was isolated from young elk which showed typical hemorrhages on the heart and lung involvement. This strain was highly pathogenic for rabbits and remained pathogenic when tested after being grown on artificial media six months later. Many strains lose their bipolar characteristics after a period of cultivation on artificial media and many lose their pathogenicity. This is not true of the Buffalo strains. A number of strains of *P. aviseptica* were pathogenic for rabbits.

Many cases of hemorrhagic septicemia are diagnosed in the field which are due to other conditions, such as toxic products in the feed. Hemorrhagic septicemia is not a very common disease, with the exception of fowl cholera.

Summary

1. Culture Buffalo B strain B. A. I. belonging to Jones' group III produces exotoxin and endotoxin.

2. A toxoid was developed by treating toxin with formalin and precipitating with acetone.

3. Toxoid, aggressin and bacterins failed to produce an immunity in rabbits sufficient to protect against injection of Buffalo B strain.

4. The various strains of the Pasteurella group are not soluble in bile, saponin, sodium oleate, cholesterol, sodium ricinoleate, or sodium desoxycholate.

5. Sodium bicarbonate and neoarsphenamine had no effect on the progress of the disease when injected intravenously.

6. Strains of hemorrhagic septicemia isolated from various animals are rarely pathogenic for rabbits except for a few strains. Those isolated from cases of fowl cholera were always pathogenic for rabbits. Hemorrhagic septicemia is not a common disease, with the exception of fowl cholera.

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Goat Milk

Because of increasing interest in the use of goat milk for its remedial qualities, it is advisable that veterinarians be informed about goats. Goats are subject to various parasites and diseases. Preventive measures should be carried out along the lines of sanitation and the avoidance of wet grazing lands. Veterinarians who have a cattle or a sheep practice can adapt themselves to the handling of diseases of goats.

According to a paper presented at a meeting of the American Chemical Society, at Washington, D. C., in 1933, it was claimed that goat's milk contained vitamins A, B, C, D and G. Dr. P. C. Knoppe, a physician writing in *Goat World*, reports that he has found that goat's milk is beneficial in obstinate cases of gastric ulcer. He has observed the cure of several cases which had been on a goat's milk regime. Other reports have been made that many babies have been found to thrive on goat's milk when other forms of food could not be digested. When infants have any allergic condition, such as asthma, eczema or hay fever, a change to goat's milk may prove beneficial. This milk is rich in minerals and has a high content of sugar and protein. It contains an easily digested form of fat. It has been found safe to use as a substitute for cow's milk. If there is any danger of brucellosis, the milk may be pasteurized.

Peace rules the day, where reason rules the mind.—Collins.

PSEUDOTUBERCULOSIS OF DEER*

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Bergey's "Manual of Determinative Bacteriology"[1] describes Corynebacteria as "slender, often slightly curved rods, with a tendency to club and pointed forms, with branching forms in old cultures. Barred, uneven staining, not acid-fast, Gram-positive, non-motile, aerobic and no endospores. Some pathogenic species produce a powerful exotoxin. Characteristic snapping motion is exhibited when cells divide."

The genus is known as the *Corynebacterium*, and is classified by Bergey under two heads: "true diphtheria organism and the 'diphtheroid' group of bacteria." The latter is then subdivided into 21 distinct species.

The diseases caused by the genus *Corynebacterium* are termed according to the animal attacked and the lesions produced. In sheep, the species is *Corynebacterium ovis* and the infection produced is caseous lymphadenitis or pseudotuberculosis. In cattle, the species is *Corynebacterium bovis*, and the infection produced is caseous lymphadenitis or pyelonephritis. In horses, the species is *Corynebacterium pseudotuberculosis*, and the infection produced is ulcerative lymphangitis, or pseudo-glanders.

LITERATURE

This organism was first isolated by Preisz and Guinard, and an article was published in 1891. Since that time, reports of the same work have been made by workers from the United States, France, Argentine, Japan, Australia, New Zealand, Germany and Chili. Shortly after Preisz and Guinard's publication, Nocard described the same organism isolated from horses, but his work was not recognized until some time later. Pallin[2] has published an interesting account of his work on epizoötic lymphangitis. The history of this organism is dealt with in a fine manner, considering the small amount of literature available on this particular subject.

Of importance in this genus is the pathogenic species, *Corynebacterium ovis* (Preisz-Nocard bacillus). This organism was classified by the Committee of the Society of American Bacteriologists as *"Corynebacterium ovis."*

BACTERIOLOGICAL CHARACTERISTICS

This organism is a slender, Gram-positive rod, measuring 0.5 to 0.6 by 1.0 to 3.0 microns. Its staining qualities are not

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dependable, as the organism tends to stain irregularly. The usual Gram stain is best suited; however, Neisser's stain also will give good results. Pallin offers methods of staining which also are suitable. The Gram-Nicolle method and the Gram-Weigert-Kuhne method are both carefully outlined by him. In a stained preparation, it is not unusual to note a few forms of the organism as club-shaped, also a few that are striated. They demonstrate no particular method of growing and will appear singly, in pairs, and frequently in groups.

Corynebacterium ovis is an aerobe and facultative anaerobe, and grows well at an optimum temperature of 37.5° C. The growth is slow at the primary stage, but yields an abundant growth after it becomes accustomed to artificial media. Blood-serum agar and egg-albumen medium[3] are ideal for the growth of this organism. Serum-agar slants produce a thin, moist, light gray and opaque, chalky growth, after the first 24 hours of incubation. At the end of 48 hours, the growth is more luxuriant, but becomes very dry, and gives a folded, serrate appearance. Due to the latter peculiarity, difficulty is sometimes met with in transferring onto other media. Egg-albumen slants produce the same typical growth, with the exception that the growth adopts a blue color, due to absorption of the indicators used. Glycerin is not a suitable medium.

Broth causes a slight sediment. Gelatin stabs are not liquefied. Bouillon produces a granular sediment and usually forms a pellicle. It is negative to indol. There is no growth produced on potato. Litmus milk is not changed. Acid is produced in dextrose.

Case Reports

During the hunting season in 1932, two deer were submitted to the laboratory for diagnosis. These deer were killed in different regions of the country within a radius of approximately 200 miles. Abscesses were found along the trachea and in the flank region and on the pleura in both cases.

Bacteriological examinations were made. Guinea pigs and rabbits were inoculated and an unidentified organism was recovered. Since that time, more work has been done on this disease and we are inclined to believe that these two cases are comparable to the following case report.

In the fall of 1936, a two-point buck deer was submitted to the laboratory for examination. Upon postmortem examination lesions were found in the subcutaneous tissue near the horns and in the region of the parotid glands. Numerous abscesses varying from 2 mm to several centimeters in size were found

on the pleura of the ribs and the lungs. Each nodule consisted of a thick wall of fibrous connective tissue, containing a thick, greenish-yellow pus, which, at a later date, became caseous and showed calcified centers. Routine laboratory examinations proved this condition was not actinomycosis or tuberculosis. Bacteriological studies were made and blood-serum and egg-albumen media were used for the growing of the organism. Carbon dioxide (CO_2) was added as a subvention to the growth. Upon examination of the cultures, a thin cretaceous colored growth, later becoming very dry and cakey, was noted. Smear examinations from the cultural material revealed a slender, Gram-positive rod, accompanied by secondary invasions of *Staphylococcus aureus* and *Staphylococcus citreus*.

Transmission Experiments

Three guinea pigs were inoculated at the time of autopsy. Two guinea pigs (145 and 146) were inoculated intraperitoneally with 0.5 cc of a 7-cm* saline inoculum of a ground-up abscess. One guinea pig (147) was inoculated with 0.5 cc of a 4-cm saline washed-off culture.

Fifteen days later, guinea pig 146 died. Multiple abscesses were found at the point of inoculation and in the testicles. All other organs appeared normal. Bacteriological cultures were made on blood-serum agar and egg-albumen media and a slender Gram-positive rod was reproduced. The growth on the media was thin and of an opaque, chalky color, which gradually ended in minute white points. No further inoculations were made from this case.

Ninety-seven days following inoculation, guinea pig 145 was destroyed. Minute nodules were centered about the point of injection. One nodule measuring approximately 3.5 cm in diameter was attached to the lower region of the left lobe of the liver. Another nodule, slightly smaller, also was located on the same lobe. Numerous small nodules were distributed throughout the entire liver and spleen. No pathological changes were noted in the other internal organs. Blood-serum agar slants were inoculated from the curdy pus, and the growth was of the same thin, opaque and chalky appearance. Smears were made from the cultural material and the slender Gram-positive rod was identified. No further inoculations were made.

Guinea pig 147, which was inoculated with a washed-off culture, died 24 days later. Typical nodules were found at the point of injection and in the liver, spleen and kidneys. A saline

*Gates nephelometer was used for all standardizations.

inoculum was made by breaking up some of the nodules in a mortar and standardizing at 4 cm. Guinea pig 132 was injected subcutaneously with 0.5 cc of this inoculum.

Guinea pig 132 died 18 days after inoculation. Typical nodules were found in the liver, spleen, kidneys and on the peritoneum. An inoculum was prepared by macerating abscesses in a mortar and standardizing at 7 cm. One-half cc was injected into guinea pig 138.

Guinea pig 138 died 16 days after inoculation. Nodules were found in the liver, spleen and on the peritoneum. The testicles were enlarged and numerous small nodules were attached. Cultures were made from the pus, and incubated at 37.5° C. for 48 hours. No further inoculations were made.

Guinea pig 140 was inoculated into the groin, with 2 cc of a washed-off culture from g.p. 147. Death occurred 17 days after inoculation, and nodules were found at the point of injection and on the peritoneum, and one-pin-point growth was found in the liver. Cultures were made and incubated 48 hours. No further inoculations were made from this case.

One rabbit was injected intraperitoneally with 2 cc of the same material as g.p. 140, but died the following day. Death was probably due to toxemia.

On January 4, guinea pigs 123 and 125 were injected with a suspension made from a pure culture, which had been in the refrigerator at 34° F. for 16 days. Guinea pig 123 was injected intravenously with 0.5 cc of this inoculum and died seven days later. Before death the eyes were secreting a purulent discharge. Cultures were made from this pus and the Preisz-Nocard bacillus was demonstrated. Nodules were found in the heart, lungs, spleen, kidneys and peritoneum. Cultures were made on blood-serum and egg-albumen agar slants.

Guinea pig 125 was injected subcutaneously with 0.5 cc of the same material as was g.p. 123. This pig died at the end of 46 days. Four days previous to death, it was noted that respiration was very uneven and difficult. Pronounced fibrillar muscular trembles also were noted at this time. A slight discharge from the eyes was observed. Cultures were inoculated from the pus enclosed in the nodules. Material used for cultural inoculations was carefully removed from the lining of the nodule as reproduction proved more satisfactory. Upon postmortem examination, a nodule measuring 3.5 cm in length and 2.5 cm in width was found at the point of inoculation. The liver and spleen showed a complete nodular involvement with practically no normal tissue remaining. Two nodules were attached to the muscle

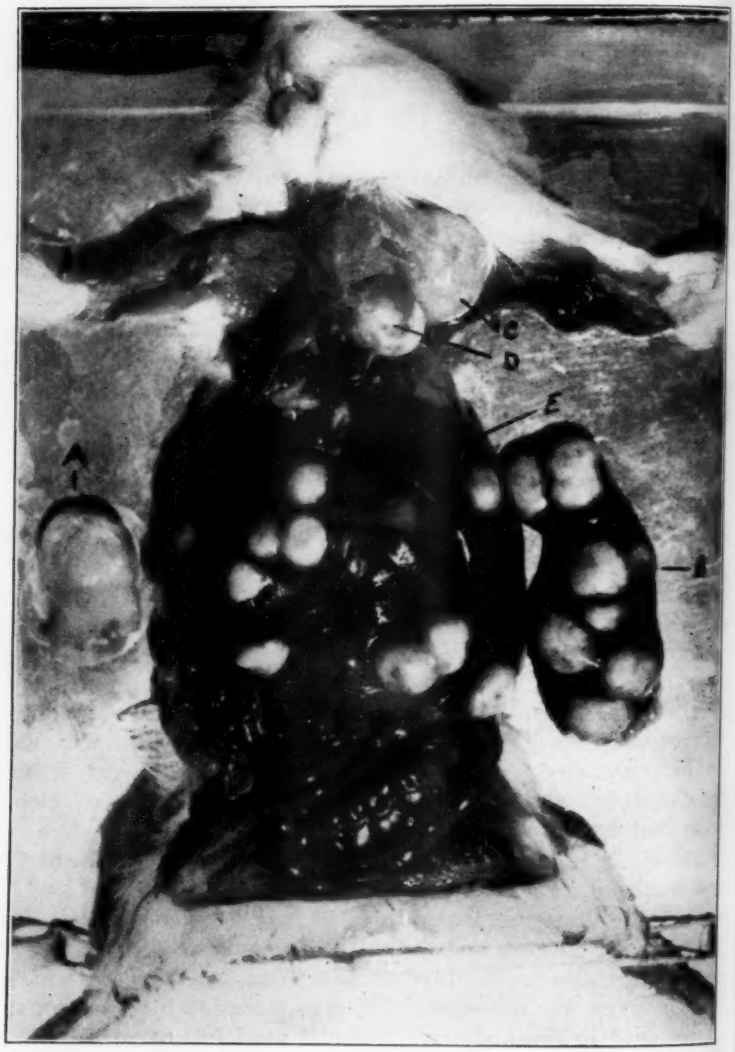

FIG. 1. Guinea pig 125. Death occurred 46 days after inoculation. A, nodule at point of injection; B, nodules in spleen; C and D, nodules attached to the muscle covering the esophagus; E, nodules in liver.

covering of the esophagus, one varying in size from 4 mm to 3 cm in diameter. No further inoculations were made from this case.

On February 12, a 4-cm suspension was made from a pure culture. A series of inoculations were made at this time from this suspension. Two chickens were inoculated into the wing vein with 0.5 cc. A Chesapeake retriever was inoculated into the saphenous vein with 2 cc. A mouse (*Mus musculus*) was inoculated intraperitoneally with 0.75 cc. The chickens and dog were destroyed 19 days after the inoculations, and no pathological changes were noted. The mouse died six days after inoculation and minute nodules were found on the pericardium. Smears revealed the Preisz-Nocard bacillus.

Two rabbits (23 and 24) were inoculated intravenously with 2 cc of the same material.

Sixteen days following inoculation, both rabbits were destroyed. Minute nodules were found in the liver and kidney. Bacteriological cultures revealed the Preisz-Nocard bacillus.

Rabbit 24 was greatly emaciated. Upon examination, the liver, kidneys and lungs showed marked nodular involvement. An extensive caseous inflammation of the subcutaneous tissue was found. This inflammatory condition practically covered the entire ventral surface of the body. Various walled-off nodules were scattered throughout this inflammation. Bacteriological cultures were made and the Preisz-Nocard bacillus was revealed.

Discussion

The distribution of the "diphtheroid" group of bacteria is of a widespread nature in the United States and in foreign countries. The disease was so common in China some years past, that a layman was capable of recognizing it. The species *Corynebacterium ovis* is reported to have occurred in bands of sheep as far back as the middle of the nineteenth century. Disease caused by the *Corynebacterium ovis* is reported to be not uncommon in sheep in the United States. However, we have never observed, and there has never been reported to the Montana Livestock Sanitary Board, lesions in Montana sheep similar in gross pathology to those observed in Montana deer.

It is of interest to note that in our experimental transmissions, it was found impossible to transmit the infection either to chicken or dog. Mice are suitable for such inoculations, as they are very susceptible to the infection caused by this organism.

It is believed that the organism lives in the ground. If that be the case, then the ground itself acts as a reservoir and becomes the source of infection. It occurs most frequently in sheep, in

the intestinal tract and in the respiratory passages. It may also enter the body through wounds and abrasions and set up a local infection, which if virulent enough, will develop into a chronic case. It is of a general opinion that the feces are an excellent means of dissemination.

It is undetermined as to how deer become infected, but it is possible the infection may be correlated with that of the sheep. This condition in deer has been called to our attention in only three cases. What the percentage of infection and the percentage of fatality in deer are in this country, we are at a loss to state. We are of the opinion, however, that this disease in deer is not of common occurrence, as the laboratories of the Montana Livestock Sanitary Board work in close contact with the State Fish and Game Commission, and disease conditions in wild animal life are immediately reported to this department.

SUMMARY

1. Eight guinea pigs, three rabbits, two chickens, one dog and one mouse were inoculated with suspensions of *Corynebacterium ovis.*

2. Guinea pigs, rabbits and mice are susceptible; apparently chickens and dogs are not susceptible to this organism (*Corynebacterium ovis*).

3. According to data, the percentage of infection is small in deer compared to that in sheep.

4. All transmission experiments and cultural and morphological characteristics classify this organism found in deer to be that of the species *Corynebacterium ovis.*

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[2]Pallin, W. A.: A Treatise on Epizoötic Lymphangitis. (London, 1904.)
[3]Loewenstein, E.: Culture medium. Ann. de l'Inst. Past., 1 (1933), p. 161.

Dog Population

According to a report in *Dog World,* approximately 590,000 dogs were licensed in Pennsylvania in 1936. Although license fees amounting to $725,000 were collected by the state, only about $32,000 was paid out for claims for animals killed by dogs.

New York City and Buffalo have systems of independently licensing their dogs. The balance of the state of New York licensed 407,000 dogs during 1936. Adding 400,000 dogs for New York City and 60,000 for Buffalo, the total for the state would be 867,000, or about one dog to every ten persons.

SYMPTOMATOLOGY OF VITAMIN-A DEFICIENCY IN DOMESTIC ANIMALS*

By G. H. HART *and* H. R. GUILBERT

Division of Animal Husbandry, College of Agriculture, University of California, Davis, Calif.

In our experimental work with vitamin A and the minimum requirements of this essential, together with field observations made during the progress of the work, we have been impressed with the variety of symptoms shown by animals as evidence of the existence of this deficiency. The explanations for some of the manifestations have been open to more than one interpretation, and this has led to some confusion in the literature regarding the etiology.

In the early stages of research following the discovery of this vitamin in 1914, the classical manifestation in the white rat became well known as xerophthalmia, which resulted from drying, due to involvement of the tear-glands, followed by keratinization of the cornea. In contrast to this drying action in rats, it is common to have manifested in domestic animals excessive lacrymation, with corneal lesions developing late or not at all. It is now known that the epithelial cell portions of structures throughout the body also may become involved. In addition to the widely distributed vulnerable epithelial cells, both the peripheral nerves and the central nervous system may show pathological alterations.

It is therefore not surprising that the variety of manifestations may be extensive. In addition to this, different animals of the same species under the same general conditions may become affected quite differently. Species variations in symptomatology are also quite marked. The situation must be viewed from its broadest aspect, or confusion in diagnosis will be common.

EYE LESIONS

The first symptom in domestic animals involves vision and consists in the development of night-blindness. It is pathognomonic of the condition but is very likely to be overlooked unless suspected and demonstrated. It is observed only in twilight or after darkness by furnishing a small amount of light from an electric globe or lantern. Animals with apparently normal vision in daylight, when moved about under these condi-

*This report is part of an investigation on the relation of nutrition to reproduction, which became coöperative with the United States Bureau of Animal Industry, July 1, 1929. Received for publication, April 8, 1937.

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tions, will run into each other, fences, supporting columns or other objects and become nervous and excitable. By arranging a maize of obstacles through which the animals are driven, the affected ones will run head-on into them, while normal animals will avoid them without difficulty even at considerable speed in the dim light.

This symptom is considered by Wald[1] to be conditioned upon a single physiological function of vitamin A. He has shown, in the normal animal, that the visual purple of the retinal rods is bleached out in bright sunlight and that vitamin A is the precursor of this substance which is essential to vision in light of diminished intensity. He has further demonstrated that visual purple, in this visual process, is bleached to retenene or visual yellow and this in turn is partly resynthesized to vitamin A. Night-blindness occurs when reserves of vitamin A are reduced to the point where insufficient is available to support this visual cycle and responds immediately to vitamin-A intake.

One form of amaurosis is definitely due to this deficiency. Once developed, it rapidly becomes a permanent condition which is not improved no matter how high or for how prolonged a period the intake of the vitamin is maintained.

It results from degenerative changes in the optic nerve. In this condition the pupils are widely dilated and there is a quite characteristic greenish coloration inside the eyeball when viewed through the cornea with the animal facing the light. Deficiency early in pregnancy, followed by adequate vitamin-A intake, may produce permanently blind but otherwise normal calves at birth. Blind pigs and pigs in which there was a complete absence of eyeballs have been produced experimentally in this manner by Hale.[2] This form of blindness has caused some confusion in the literature because it has occurred under conditions in which vitamin-A deficiency was not suspected and resulted in other possible causes being suggested.

Crocker[3] reported blindness in newborn calves caused by what he termed insidious rachitis. There were 16 cases in Guernseys on two farms in Pennsylvania. No external change was present in the eyes. The dams were all on high concentrate and low roughage feeding for intensive production. On postmortem the affected calves showed stenosis of the optic canal with chronic optic neuritis, and the condition was diagnosed as insidious rachitis with deformation of the sphenoid bone.

de Schweinitz[4] and de Schweinitz and De Long[5] have published on a similar condition in calves from Guernsey cows under the

name of papilledema or choked disc, and suggested that the defect may be hereditary. A paper by Moore, Huffman and Duncan[6] describes 24 cases in calves and growing dairy animals, of a type of nutritional blindness which the authors state apparently differs from true vitamin-A blindness. In their cases the loss of sight without inflammation of the external eye structure was associated with weakness, spasms and paralysis. Degeneration of nerve tissue, including that of the optic nerve, is well established in vitamin-A deficiency. All the cases reported by the workers mentioned above appear to us to be manifestations of vitamin-A deficiency. Since this defective sight apparently can be corrected occasionally if vitamin A therapy is started in time, evidently the stenosis of the optic canal is secondary to degenerative changes and to atrophy of the optic nerve.

In our heifer 542, complete blindness developed in daylight with dilated pupils which we considered evidence of optic nerve involvement. Seventeen days after this condition was manifested, vitamin A was supplied in ample amount. During the second month of ample vitamin-A intake, sight apparently returned to normal in daylight. Recovery was not complete and this could be detected by permanent defective vision in dim light that could be confused with night-blindness. In sheep, following the development of true night blindness, many cases quickly develop a partial impairment of vision detectable only in dim light which is permanent regardless of vitamin-A therapy.

Further confirming evidence that stenosis of the optic canal is secondary has been obtained with three of our test animals that became blind, without showing external lesions in the eyeballs, during vitamin-A privation. Photographs of the dissected optic nerves of these animals are shown in figure 1. "A" is from a calf which was born blind and was autopsied at 2½ months of age. Its mother was deficient in vitamin A during the fifth month of pregnancy and then sufficiently supplemented for the remainder of the gestation. Marked degeneration of the optic nerves, where they pass through the optic canals, is shown. The bony canals showed conspicuous stenosis. "B" is from calf 570, which became totally blind at eight months of age on a level of vitamin-A intake that had previously restored normal vision in semi-darkness. It was given 5 cc of cod-liver oil daily, which supplied about four times the minimum level for a period of four months without improvement. At the end of this time, it was slaughtered. Optic nerve atrophy had occurred in the same region. "C" is from bull 543, which became blind at eight months of age and was slaughtered at 32 months of age. In this long-

standing case, the atrophy is also most conspicuous in the section of the nerves that pass through the optic canals. Shrinkage, however, is plainly evident through a much larger part of the nerves and this is particularly manifest in the nerve on the left side of the illustration.

The constriction of the bony canals was less conspicuous in the last two cases than in the first one, probably because the animals were older at the time the nerve atrophy occurred. We consider this as evidence that nerve atrophy is the primary condition.

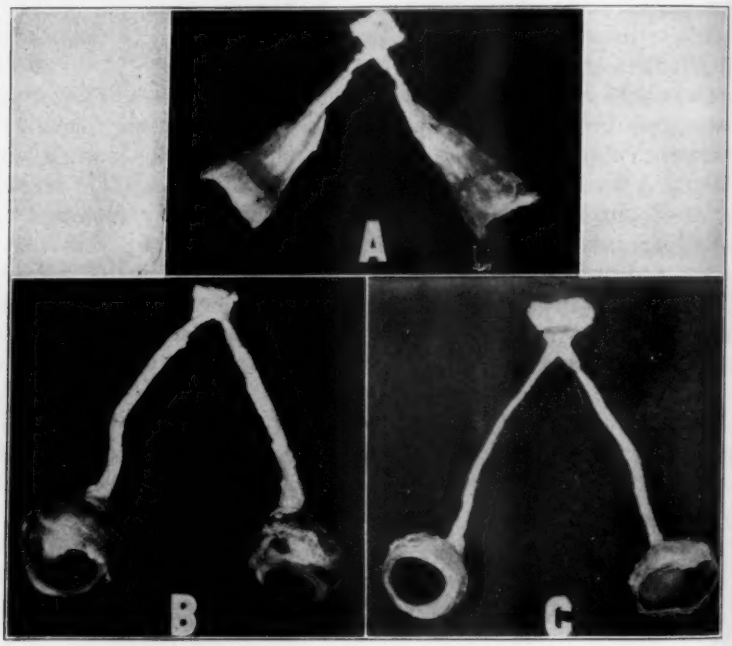

FIG. 1. Photographs of the dissected optic nerves of animals that became blind during vitamin-A privation.

Kulman *et al*[7] report both of these types of blindness in their test calves fed a low-vitamin-A diet consisting of beet pulp, cottonseed meal and bone meal. The blindness without visible change in the eyeball, except dilated pupil, was permanent and occurred simultaneously or independently of the xerophthalmia cases.

Ulceration of the cornea from lack of vitamin A is one of its manifestations and is most commonly found in the dog, but also in other species. It is associated with bacterial invasion, which is secondary.

Nervous Symptoms

These may result from alterations within the central nervous system or in the peripheral nerves. The latter are quite similar to what has been described in the optic nerve and the symptoms will depend on the nerves affected. Hughes, Aubel and Leinhardt[8] found degenerated nerve bundles in the optic thalamus, optic, femoral and sciatic nerves and also in certain areas in the spinal cord of swine. No cerebral lesions were found but symptoms suggested impairment of this tissue.

Zimmerman and Cowgill[9] have recently published on lesions of the nervous system in this deficiency in the white rat. They found in all cases the myelin degeneration in the sciatic nerves was greater than in the brachial plexus. No lesions were demonstrable in the cerebrum by any of their staining methods. In the spinal cord degeneration of the medullary sheaths in both sensory and motor tracts was quite extensive.

One form of posterior paralysis in hogs is due to this cause. In one group of Poland China hogs in the experiments of Guilbert, Miller and Hughes,[10] at this station, the posterior paralysis developed before night-blindness could be demonstrated. Its development is usually secondary to night-blindness. Prior to the development of paralysis, there is incoördination of movements. Back muscles on one side only may become involved, resulting in more or less marked scoliosis. Vitamin-A therapy, instituted early, results in marked improvement of general muscular incoördination, weakness and even posterior paralysis, but complete recovery is rare.

The lesions in the nerves once developed are usually permanent and probably often progressive. Leg paralysis in cattle is not common but does occur and has developed in experiment animals in the front quarters. Convulsions in cattle and hogs are a peculiar form of manifestation of this condition, the pathology of which has not been studied. Animals in the early stages of the development of this symptom will go down after more or less excitement, prior to which lack of coördination of voluntary movements will be marked. In the more advanced stage, merely startling the animal by suddenly jumping toward it, or clapping the hands, will cause it to develop a convulsion, drop to the ground and stretch out with its muscles spastically contracted as though stunned with a sledge hammer. In a short time relaxation occurs and the animal will lie normally for a few moments and rise, only to repeat the performance under proper stimulus. Later, convulsive seizures may occur spontane-

ously. Various injuries may be sustained by the sudden involuntary fall, such as shelling off the horn, and bruising the nose or jaws. A condition identical with that which we have produced experimentally has been observed in steers in feed-lot toward the end of a long feeding period where the ration has been largely concentrates other than yellow corn with limited amounts of bean straw or grain hay for roughage. Death may occur in these convulsive seizures before other evidence except night-blindness has been manifested, particularly in pigs.

Involvement of Other Epithelial Structures

Here is where the greatest variety of manifestations may occur, some of which may readily be confused with conditions of entirely different etiology. The gastro-intestinal tract is lined throughout with epithelial cells. In vitamin-A-deficient poultry quite characteristic lesions are found, particularly in the mouth and throat. Beach,[11] in his original report elucidating this condition, described them as follows:

> The lesions in the mouth, pharynx, esophagus, and crop consist of collections of white caseous material in the mucous glands. On the surface of the mucous membrane they appear as circular white pustule-like caseous patches, from 0.5 to 2 mm in diameter. The application of pressure around them forces out a slender cylindrical caseous mass 2 to 3 mm in length. The number of pustules may remain small or become so large as to almost cover the mucous membrane.

In cattle, sheep and hogs gastro-intestinal involvement is associated with diarrhea. With steers in feed-lot this may be the first symptom noted by the feeder although the unsuspected night-blindness will have existed for some time previously. In newborn calves excessively loose bowels from this cause may confuse the condition with white scours. Vitamin-A therapy will quickly relieve the condition if caused from this deficiency, while no amount of vitamin A will of itself prevent or cure white scours.

In the respiratory tract lung abscess is not uncommon. It was found at postmortem examination in one of our experiment cows. In growing calves under natural conditions with subminimum intake, lung involvement becomes a progressive pneumonia and often the final cause of death. Such cases on postmortem show pneumonic areas interspersed with soft nodules the size of a pea to a marble which contain air and masses of pus cells.

In the genital tract changes also occur but cows will come in heat in advanced stages of the deficiency when having convulsive seizures. One of our bulls (541) died of the deficiency and no sperm were found in the testes. On the other hand, bull 543,

previously mentioned, was so badly deficient that he became permanently blind and had frequent convulsions but later bred and successfully impregnated six cows after all evidence of deficiency had been removed except blindness through ample vitamin-A intake.

Much experimental data justify the statement that the genital-tract changes in vitamin-A deficiency in the male are temporary, while those caused in this sex by vitamin-E deficiency are permanent. In the pregnant females, vitamin A does not pass the placenta much faster than it is utilized by the growing fetus. Even though the pregnant mother has ample storage of it, the offspring is born with a small amount of storage in the liver. If storage in the mother and content in her feed are low, sufficient of the substance may not reach the fetus to keep it alive and abortion results. This condition may readily be confused with infectious abortion and requires blood-testing of the dam and chemical examination of the liver tissue of the fetus to make a differential diagnosis.

Mason[12] found in the rat that fetal death resulted from disturbances in the nutritive supply of the fetus originating from pathological alterations in the epithelial cells of the maternal placenta, consisting of focal necrosis in the maternal decidua adjacent to the fetal trophoblast. This always preceded alterations in the fetal tissue and the latter consisted of general disintegration from nutrition being affected by the placental injury. On the other hand, in vitamin-E deficient rats, receiving adequate vitamin A, there was early degeneration of fetal tissues followed later by changes in the decidua.

Mason and Ellison[13] suggest that vitamin A plays a part in protein metabolism or synthesis within the epithelial cell. Thus, in cells deprived of vitamin A, the production of glycoproteins or mucin apparently are retarded and albumenoids or keratin increased. Their studies did not make possible a differentiation between the keratinizing of vaginal epithelium in vitamin-A deficiency from that normally occurring in the rat and other species at estrus under the influence of hormonal factors.

When the deficiency exists on the range, many calves and lambs born at term and alive will die during the first few days of life because they are too weak to stand and nurse or have severe diarrhea. Colostrum is a rich source of this substance for the newborn, provided the mother is not too greatly depleted.

When the genito-urinary system is involved, the lesions are mainly limited to the kidneys. Parenchymatous degeneration of the epithelium lining the tubules may occur and the so-called

large white kidney result. It has been suggested that kidney lesions may have been associated with the few cases of edema of the legs, which is occasionally seen in affected animals. From experimental evidence collected by Moore and Hallman,[14] these investigators suggest that the well-known white spotted kidney in calves may result from vitamin-A deficiency. In poultry marked accumulation of urates occurs in the renal tubules, appearing as a network of fine white lines. The ureters may be distended with them and there may be deposits in other body tissues.

It is thus seen that lack of this single dietary essential may cause a wide variety of alterations in body tissues and symptomatology. Under conditions of drouth or unseasonable weather, such as occurred in northern California this past winter, with no early rains and a very severe cold winter preventing growth of green feed, losses from this condition become of real economic importance. On the other hand, its comparatively rare appearance under more stable feeding conditions results in confusion in diagnosis unless feeding regimen is taken into consideration.

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History is the essence of innumerable biographies.—Carlyle.

SULFUR IN THE CONTROL OF EXTERNAL PARASITES OF CHICKENS

Preliminary Report*

By M. W. EMMEL, *Gainesville, Florida*
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For many years it has been the opinion of investigators, including the author, that sulfur fed to animals in the feed becomes a relatively inert substance in the intestinal tract and is subsequently eliminated practically unchanged in the droppings of animals to which it is fed. Many poultrymen, however, have recited to the author experiences in which the feeding of sulfur to chickens produced a beneficial effect in the control of lice.

One poultryman was so insistant that feeding sulphur to chickens would control lice that six Barred Rock hens heavily infested with lice (*Menopon stramineum* and *Menopen gallinae*) were placed in a small house with wire floor and wire sun-porch and fed a commercial laying mash to which 5 per cent sulfur (commercial flour) had been added. When examined at the end of one week, a number of dead lice were found among the feathers and the infestation was considerably reduced as compared with four control birds from the same flock housed under similar conditions, but receiving no sulfur. At the end of two weeks, lice were difficult to find on the sulfur-fed birds, while at the end of the third week, no living lice could be detected during a careful examination. The infestation of lice on the four control birds did not show visible reduction.

The most interesting observation was that when the feathers of the sulfur-fed birds were ruffled back, a distinct odor of sulfur dioxide could easily be detected. Although faintly present at the end of one week, this odor reached its maximum after about three weeks of sulfur feeding.

The experiment was repeated with eight Rhode Island Red hens placed in a hen battery inside a building. At the end of three weeks, the odor of sulfur dioxide could be but faintly detected if at all; the infestation of lice was reduced about 25 per cent as compared with the four control birds receiving no sulfur. The birds were then placed in outside cages from which the birds had access to sunshine. In two days, the odor of sulfur dioxide could be detected easily on the skin surface and among the feathers of all of the sulfur-fed birds. Complete

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control of lice was recorded in the sulfur-fed birds at the end of a week. The infestation in the control birds did not show visible reduction.

The two experiments were repeated with identical results. Since dusting with sulfur controlled infestations of lice, precautions were taken to eliminate this factor.

Five per cent sulfur has been fed continuously to two groups of four Rhode Island Red hens for three and ten months, respectively. After a period of about six to eight weeks of continued sulfur feeding, the odor of sulfur dioxide in general is not so pronounced and implanted infestations of lice are controlled much more slowly than the original infestation. This would seem to indicate that intermittent feeding of sulfur would perhaps be more effective in controlling lice in chickens than continuous feeding over long periods of time.

Herrick and Holmes[1] reported that birds receiving 5 per cent sulfur in their feed for the control of coccidiosis often showed an irritated cloaca. This condition was not observed by this author. It was noted, however, that the skin of sulfur-fed birds became markedly scaly; considerable epithelial débris was observed. In some instances the condition was reduced in intensity with continued feeding. A "puffed" appearance often developed around the eyes. The surface of the comb and wattles became scaly, giving them a grayish cast.

These experiments would indicate that at least a small portion of the sulfur fed was absorbed from the intestinal tract in some form and eliminated by way of the skin. Since the element sulfur is practically odorless, oxidation of the sulfur to sulfur dioxide must apparently occur. Since the experiments show that the treatment is more effective when sulfur-fed birds had access to sunshine, it would appear that sulfur dioxide is the agent responsible for the destruction of lice. The mechanism of elimination and the form in which sulfur is eliminated from the skin of sulfur-fed birds has not as yet been determined.

Two experiments were conducted in which 5 per cent sulfur was fed to hens heavily infested with fleas (*Echidnophaga gallinacea*). This treatment was augmented by sulfurizing the yards at the rate of 100 pounds of sulfur per 400 square feet of area and placing a light coating of sulfur on the floor of the houses under the litter. The sulfur was scattered over the ground and mixed with the top soil with a rake. After several days, the fleas began to drop from the birds. At the end of three weeks, the infestations were under complete control; the feeding of sulfur

was discontinued. Although a period of four months has elapsed in each instance, reinfestation has not occurred.

The owner of a lawn heavily infested with fleas (*Ctenocephalus canis*) appealed to the author for assistance in eradication. One hundred pounds of sulfur was dusted on the lawn (75 x 75 feet). complete eradication was accomplished almost immediately and remained so except for several days following a rain two weeks after the application of sulfur. Three hundred pounds of lime was applied to the lawn two months after being sulfurized, since lime counteracts acidity caused by the application of sulfur. Complete eradication was accomplished with no injurious effects to the lawn.

The owners of small animals often have basements which become infested with fleas. It occurs to the author that dusting with sulfur would be an effective means of eradication.

It has been found that direct contact with sulfur kills an adult flea in from five to ten minutes at room temperature; when adult fleas were subject to fumes arising from sulfur at room temperature (fleas one inch above the sulfur), death occurred in several hours. An incubator temperature of 37° C. hastened death in both instances.

The same procedure as was used in the instance of fleas was given a trial on two tick-infested (*Argas persicus*) flocks. The infestation on the birds was reduced but not so rapidly as in the case of fleas. Reinfestation of the birds did not occur after the birds became tick-free.

Direct contact with sulfur at room temperature failed to kill the adult tick. It is thought that sulfurization of the soil increased the soil acidity, creating an unfavorable environment for the development of young forms of parasite. Since considerable free sulfur exists in the soil for some time after sulfurization, it may be possible also that direct contact with sulfur may be a factor.

Sulfur has been found to be effective in controlling the mite, *Dermanyssus gallinae*, in three infested poultry-houses. The houses were cleaned mechanically as thoroughly as possible. Sulfur was placed on the floors, dropping-boards and nests to a depth of perhaps 1/16 of an inch. A broom was used to work the sulfur into the cracks. In each instance the mites began to disappear in several days and, at the end of a week, control seemed to be complete. In one instance in which soil served as the poultry-house floor, sulfurization proved effective on heavily mite-infested premises. In another instance merely dusting sulfur on the litter and under the nesting material proved as effective in control as the first method described.

Since many phases of these experiments have given results contrary to the general prevailing opinion, particularly in regards to the feeding of sulfur to chickens, further experiments are in progress.

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Phi Zeta Initiation at Iowa State College

The Iowa State College chapter of Phi Zeta, national veterinary honor society, held its annual initiation ceremonies in the Memorial Union, at Ames, May 26, 1937. Following the initiation, a banquet was served to 42 members. Dr. S. H. McNutt, president of the local chapter, presided as toastmaster. The address of the evening was given by Dr. R. E. Buchanan, Dean of the Graduate College and Director of the Agricultural Experiment Station.

Two honorary members were initiated: Dr. W. A. Aitken (I. S. C. '18), of Merrill, Iowa, and Dr. J. C. Carey (I. S. C. '19), of West Liberty, Iowa, president of the Iowa Veternary Medical Association. The following active members were initiated: Dr. W. R. Anderson (I. S. C. '30) from the faculty; L. A. Dykstra, D. R. Cordy, C. G. Hanna, L. A. Bowstead, B. J. Sinkler, W. E. Brown, R. N. Brenny and W. S. Monlux, from the senior class, and V. U. Thomson, C. I. Angstrom, L. M. Griffin, C. E. Guthrie and N. D. Crandall, from the junior class.

Drug Seizures

Among the drugs and medicinal preparations seized by the U. S. Food and Drug Administration recently were 17 cans of impure ether; a shipment of "Cereal Lactic," represented as a good source of *Bacillus acidophilus*, whereas the product was markedly deficient in that organism and contaminated with others; "Effervescent Seltzer," containing only 5 grain of acetanilid per ounce although labeled to contain four times that amount; "Geno Tablets," for bowel trouble in chicks, intestinal disorders in all ages of poultry, coccidiosis and typhoid cholera; and "Williams Turkey Tonic," a solution of hydrochloric acid, Epsom salt, an iron salt and a plant extract, for amebic infection and other ailments of turkeys. The last-named product was sold in violation of the federal Caustic Poison Act, by reason of the fact that the word "Poison" was not of the proper size, and the antidote given was not complete.

THE TOXICITY OF CROTALARIA RETUSA L. SEEDS FOR THE DOMESTIC FOWL*

By M. W. EMMEL, *Gainesville, Fla.*
Florida Agricultural Experiment Station

Five species of *Crotalaria* have been reported by a number of investigators to be toxic for various species of animals: *Crotalaria sagitallis* L. (horses), *C. burkeana* Benth (cattle), *C. juncea* L. (sheep), *C. dura* Wood and Evans (horses), and *C. spectabilis* Roth (cattle, hogs, horses, sheep and chickens as well as in force-fed turkeys, dove and quail).

It has not been established that *C. retusa* L. is toxic. Becker, Neal, Arnold and Shealy[1] observed that cattle having access to this species of crotalaria during one grazing trial did not eat the green forage of this plant. Rusoff and Neal[2] isolated at least two alkaloids from the seeds of *C. retusa* L. Both alkaloids had different melting points; one was fat-soluble while the other was water-soluble; no tests on their toxicity were conducted.

Fifteen Single Comb White Leghorn hens were force-fed *C. retusa* L. seeds. A single dosage of 100 seeds proved fatal for four birds within 14 days. Two hundred seeds force-fed in a single dose to four birds induced death within ten days. Chronic cases of poisoning were induced in the remaining seven birds by force-feeding ten seeds every other day; death occurred in from 18 to 40 days. Five birds were fed a grain mixture containing seeds of *C. retusa* L. scattered on the ground once a day. This grain mixture was readily consumed by all of the birds; death from *C. retusa* L. poisoning occurred in 15 to 32 days.

The most marked and constant gross lesion was ascites, with an accumulation of a gelatinous mass which embedded the liver and which was rather firmly adherent to this organ. Necrotic enteritis prevailed in all of the birds. Congestion of the visceral organs was observed in the more acute cases, while in the chronic type of poisoning the organs appeared pale. Innumerable pete-

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chiae on the serous membranes and a "marbled" liver so characteristic of acute *C. spectabilis* poisoning were not observed. Hemorrhagic lesions, however, were often present, particularly in the more acute cases. Other lesions, both gross and microscopic, were quite similar to this type of poisoning. Edema and cloudy swelling dominated the histopathology. Anemia often accompanied by leukopenia, with a corresponding decrease in the hemoglobin reading, often were observed in the chronic cases. Differential blood counts were not made.

The toxic principle in *C. retusa* L. seeds is probably different from that of *C. spectabilis* Roth. This opinion is based on the absence of extensive hemorrhagic lesions and of such acute cases of poisoning as observed in *C. spectabilis* Roth poisoning. The toxicity of *C. retusa* L. seeds, however, for the domestic fowl is almost as great as *C. spectabilis* Roth seeds which are considered highly toxic.

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AN ERYSIPELAS OUTBREAK IN TURKEYS*

By D. E. MADSEN, *Logan, Utah*
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Beaudette and Hudson[1] have reported extensive loss in a flock of turkeys as a result of infection with the swine erysipelas organism. The following report may place further emphasis on the economic importance of this disease in turkeys and its possible relationship to sheep erysipelas infections.

A rather heavy loss among a flock of 5½-month-old turkeys located near Fairview, Utah, was investigated by the author in November, 1936. Three birds brought to the laboratory for examination were in good flesh and on autopsy revealed a catarrhal enteritis, with accumulation of mucoid, gelatinous material. The posterior intestine was filled with watery, greenish feces. There were hemorrhages on the endocardium and pericardial fat. No careful search was made for hemorrhages in the muscles, and the autopsy report does not make mention of such hemorrhages. It is possible they were present and were overlooked....

Cultures prepared from all three birds revealed small, delicate colonies, stained preparations of which showed a small Gram-

*Contribution from the Department of Animal Pathology, Utah Agricultural Experiment Station. Authorized by the Director. Received for publicaion, June 21, 1937.

positive rod with all of the characteristics of the swine erysipelas bacilus. Carbohydrate media tests also placed the organism in this group. When injected intramuscularly into two pigeons, this organism caused their death in 48 hours and one adult turkey succumbed 60 hours following subcutaneous exposure. Two chickens were inoculated but remained healthy.

The same Gram-positive rod was recovered from the pigeons and turkey. Autopsy of the turkey revealed hemorrhagic blotches over the pectoral and thigh muscles and on the pleural surface of the brisket. The liver and spleen were swollen and mottled. A few hemorrhages were located along the mucosa of the alimentary tract, on the endocardium and on the pericardial fat.

Two chickens were inoculated with known *Erysipelothrix rhusiopathiae* organisms and later serum from them was collected which agglutinated in 1:200 dilution the organisms isolated from the turkeys.

History of Outbreak

The owner had a total of 3,000 turkey poults divided into three flocks, each flock being ranged in separate areas (one-half to one mile apart). Losses occurred in only one flock of 1,300 poults. This flock was ranged near the base of a canyon on ground not previously occupied by fowl. About one mile up this canyon, were located some lambing corrals where several hundred ewes had lambed during the lambing season immediately past and for several previous seasons.

In July, 1936, there was a heavy rain storm which caused a great rush of water to come down the canyon, carrying with it large quantities of manure from the corral and depositing it over the general area occupied by the 1,300-poult flock. Again, in August, another rain storm washed large quantities of manure over the same area. Efforts to obtain any information regarding the presence of infectious arthritis in the sheep which had occupied the corral have to date been unsuccessful.

Utah lambs are not infrequently affected with this disease, especially where sheepmen have used the same lambing corrals for several years. It is interesting to note that the turkey losses were confined to the flock exposed to the accumulation washed down from the lambing corral. The owner of the turkeys stated that in previous years he has brooded his turkeys in an old lambing-shed and ranged them over old sheep feeding grounds with no disease loss. This, however, was in a different location from the site above mentioned and was occupied by a different flock of sheep.

In the outbreak here reported, there was a loss of 325 birds, or 25 per cent of the flock. The loss began on November 12, when the poults were 5½ months of age. Losses were heavy for two weeks and then gradually tapered off until all loss subsided four weeks after the outbreak. Of the 325 turkeys lost, only 25 were females. It was reported that a much higher percentage of the flock was affected than succumbed. A higher percentage of the hens recovered than did toms. The badly affected birds were off feed, crouched down, and remained aloof from the remainder of the flock. Only a few showed any evidence of diarrhea. Usually death followed in a few hours after pronounced depressed symptoms were noted. The tubular leader (caruncul) on many of the recovered birds dried up and dropped off.

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TRAUMATIC PERICARDITIS*

By EMIL E. GRINSTEAD, *Yakima, Wash.*

This case is reported to show that acetonemia was a secondary factor in traumatic pericarditis.

On June 3, 1934, I was called to examine a grade Holstein cow, aged three years. This cow had calved one month before being examined. The owner had noticed that the animal was gradually losing her appetite and was rapidly becoming emaciated. Within the past 15 days, the milk-flow had diminished 75 per cent. The cow was salivating slightly and grinding her teeth periodically.

The bowel movements were scanty, and atony of the rumen was noted, with decreased peristaltic movements of the intestines. The cow was standing with her back arched and head lowered. The heart-beat and temperature were normal. There was no stiffness of the fore quarters in evidence. No pain was induced or jugular pulse noted by kneading with the fist over the region of the heart or xiphoid cartilage. The urine was withdrawn and it gave a positive reaction to a test for acetonemia.

TREATMENT

An intravenous injection of 200 cc of a 50 per cent solution of glucose was given, and one rumen tablet every four hours

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during the day. The urine test was negative 24 hours later. There was a slight increase in the appetite, bowel movements and milk-flow. This cow died three days later and, upon postmorten examination, a four-inch wire was found protruding through the reticulum, diaphragm, pericardial sac, and into the heart. No abnormal fluid or pus was found in the pericardial sac. The heart had a cauliflower growth at the apex, which measured two inches in length and diameter.

PULMONARY COCCIDIOIDAL GRANULOMA

A New Site of Infection in Cattle*

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Since bovine coccidioidal granuloma was first reported by Giltner[1] in 1918, a total of 22 cases in animals has been recorded in veterinary literature up to 1935. Beck, Traum and Harrington[2] reported 19 bovine cases and one case in a sheep. Two of the authors encountered two bovine cases, one occurring in Colorado[3] and the other originating in the Southwest.[4] Aside from the latter two cases, the disease was found in animals raised in California.

Although the incidence of the disease in animals showed no marked increase from 1918 to 1935, the *Weekly Bulletin* of the California State Department of Health[5] reveals a rather conspicuous increase in the number of human cases of coccidioidal granuloma in California. From the period June 1, 1931, to July 1, 1936, the number of cases in man increased from 264 to 450. Of the total number of human cases reported, 224 (50 per cent) were fatal.

A survey conducted by the California State Department of Health[6] to determine the incidence and geographic distribution of coccidioidal granuloma in man and animals showed the disease to be confined to the central and southern sections of the state.

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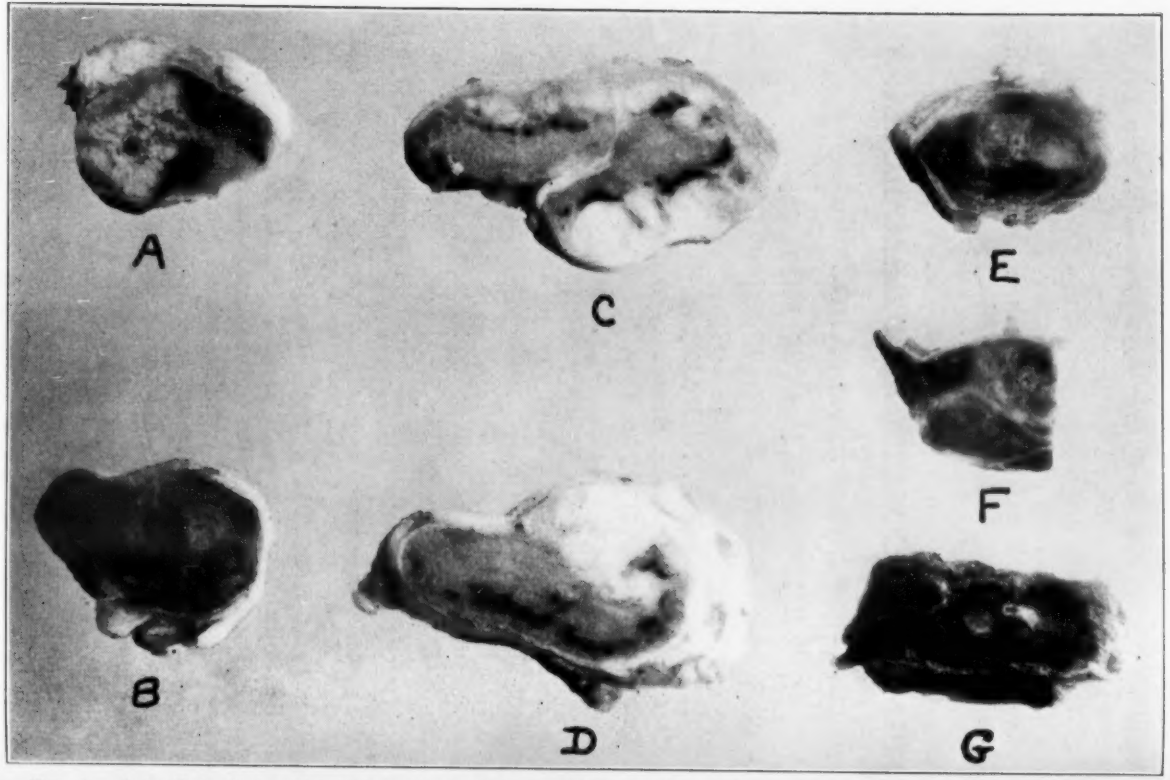

FIG. 1. A to E inclusive show lymph-node lesions. Natural size. Note the variation in size of the granulomatous foci and the striking resemblance to tuberculous lesions; F and G are lung lesions. Note the encapsulation of the foci in F.

While the disease in man frequently becomes generalized, affecting practically all tissues and organs, the lesions heretofore observed in naturally infected animals have been confined to the bronchial and mediastinal lymph-nodes. These animals exhibited no apparent clinical symptoms and were found to be affected when slaughtered for food.

It is the purpose of this paper to report 47 additional cases of bovine coccidioidal granuloma and the finding for the first time of pulmonary lesions in cattle.

Case Reports

Lot 1: On May 4, 1937, 128 Hereford cattle, approximately six years of age and in fair condition, were slaughtered for food purposes in Los Angeles. Upon postmortem inspection conducted by Dr. C. F. Kellogg, of the Meat Inspection Division, U.S.B.A.I., a total of 42 animals in the lot showed lesions suspected of being coccidioidal granuloma. The character of the lesions varied somewhat in size and consistence. Calcareous deposits were not detected. Some of the lymph-nodes were suppurative, containing a creamy, tenacious pus and strongly resembled actinomycotic pus; while other lesions in the lymph-nodes and lungs were of a cheesy consistence, resembling somewhat the caseous lesion of tuberculosis (fig. 1). The lesions were noted as affecting the following tissues: bronchial lymph-node only in two cases; 27 animals showed mediastinal lymph-node lesions only; seven cases showed bronchial and mediastinal lymph-node involvement, and six animals revealed mediastinal and slight lung lesions.

Lot 2: On May 14, 1937, 61 Hereford cows about six years of age were shipped to Los Angeles for slaughter. Dr. H. L. Kilhoffer, also of the federal Meat Inspection Division, found five of the lot showing lesions in the bronchial and mediastinal lymph-nodes similar in character to those observed in the first lot. Pulmonary lesions were not present in any of the five cases.

Both lots of cattle were traced and it was found that they came from two different feed-lots near Bakersfield, Kern County, California.

Representative lesions from several cases in both lots were submitted to the Denver branch laboratory of the Pathological Division for diagnosis.

Laboratory Findings

Cover-slip preparations of the purulent contents of the affected lymph-nodes disclosed many spherical bodies with a refractile double-contoured capsule. The parasites varied in size from 10 to 50 microns. Both sporulating and non-sporulating forms were

matous foci and the striking resemblance to tuberculous lesions; *F* and *G* are lung lesions. Note the encapsulation of the focal in *F*.

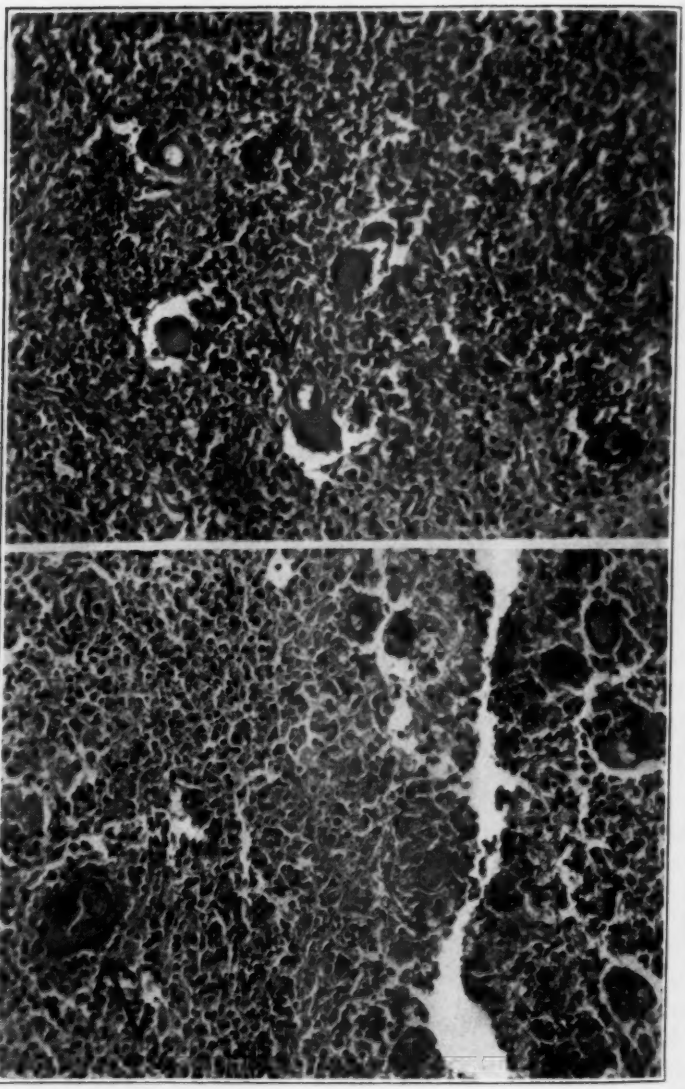

FIG. 2 (above). Several giant-cells in the field, with one enclosing a parasite showing the double-contoured capsule. Arrow points to parasite (x 150).

FIG. 3 (below). Another field showing the parasites. Several are within the giant-cells and one spherule is free in the tissue. Arrow points to one parasite in the process of rupturing (x 150).

seen. The pus from several lesions was cultured on meat-infusion agar and uncontaminated cultures of *Coccidioides immitis* were obtained from each gland after incubation for 72 hours at 37° C. The mold on solid media produced white, cotton-like colonies. Hanging-drop preparations from the growth showed an intricate network of mycelia. In the tissues, however, the organisms appeared only in the spherical form.

Histologic sections from several lymph-node and lung lesions stained with hematoxylin-eosin showed granulomatous foci consisting of connective tissue, numerous blood-vessels, lymphocytes, plasma cells, mononuclear and polymorphonuclear leukocytes, a few eosinophiles, and many giant-cells. The foci were surrounded by a connective tissue capsule. Scattered throughout the inflammatory areas were many double-contoured spherical bodies, the majority of which had been taken up by giant-cells (fig. 2). Scattered purulent areas containing spherical forms were seen within the granulation areas. Many of the parasites showed a strong affinity for eosin. An occasional spherule showed a ruptured wall. It was noted in the sections that the organisms in the process of rupturing stained deeply with eosin. This suggests to us that these particular bodies are the more recent sporulating forms of the parasite. In some fields the inflammatory reaction resembled tubercle formations and only the presence of spherical bodies within the giant-cells enabled a differentiation from a tuberculous process. The parasites were Gram-negative.

Discussion

Prior to our findings, research workers interested in animal coccidioidal grauloma have expressed the belief that lesions of the disease do occur in animals elsewhere than in the thoracic lymph-nodes. In the two cases previously reported by the authors, the animals were under two years of age and may have been slaughtered before the lymph-node lesions had an opportunity to progress. However, the age of the animals which showed lung lesions was approximately six years. It would appear then that if animals affected with the disease were permitted to live longer, the probability of finding lesions of wider dissemination would increase.

In view of the frequent skin localization of coccidioidal granuloma in man, Dr. J. Traum, of the Division of Veterinary Science, University of California, in his studies of the so-called "skin lesions" of bovine tuberculin reactors, made a diligent search for coccidioidal infection in such lesions. By culture and animal inoculation methods several hundred bovine cutaneous

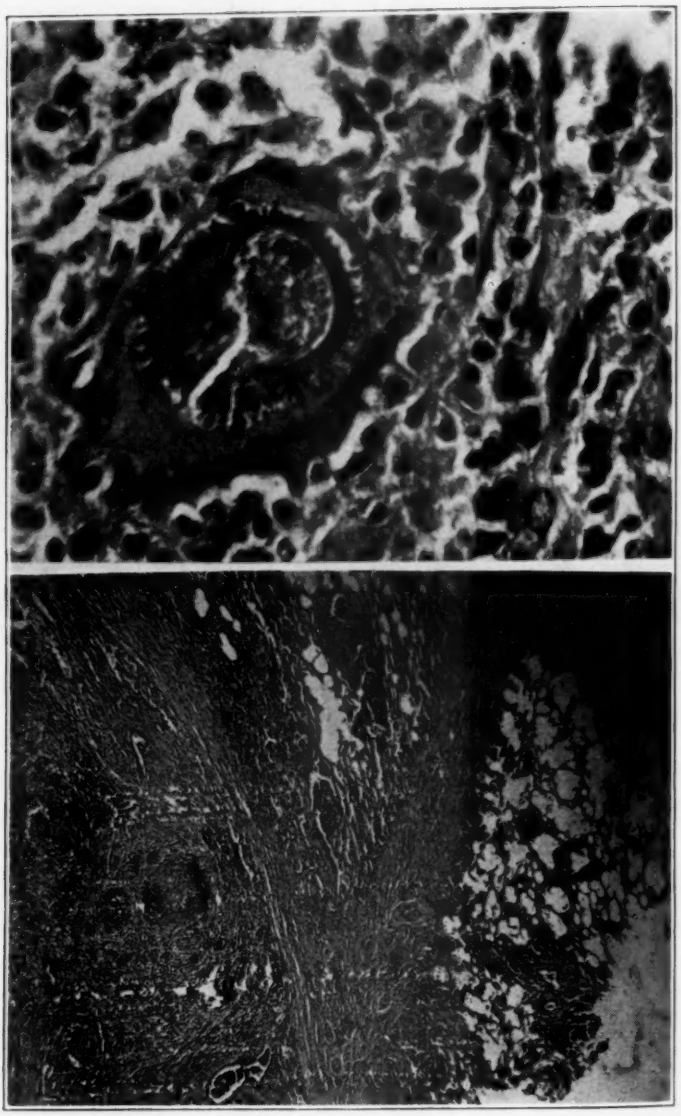

FIG. 4 (above). Higher magnification of ruptured spherule seen in B (x 550).

FIG. 5 (below). Lung lesion separated from normal parenchyma by a broad connective tissue capsule (x 15).

and subcutaneous skin nodules were found negative for *Coccidioides immitis*. Perhaps further studies of skin lesions found in cattle coming from known infected areas in California may eventually reveal coccidioidal infections.

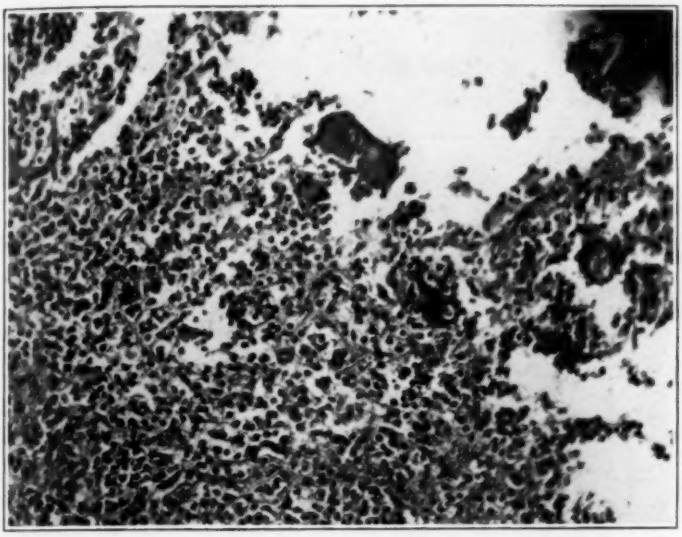

FIG. 6. Section of lung lesion showing several giant-cells containing the parasite (x 150).

SUMMARY

1. Pulmonary lesions in bovine coccidioidal granuloma are reported for the first time.

2. Forty-seven new bovine cases are here reported.

3. A survey of additional cases of coccidioidal granuloma in both man and animals continues to show a decided concentration of the disease in the San Joaquin Valley in California.

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[4]Stiles, G. W., Jr., and Davis, C. L.: A case of bovine coccidioidal granuloma from the Southwest. Jour. A. V. M. A., lxxxvii (1935), n. s. 40 (5), pp. 582-585.

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[6]Calif. Dept. Pub. Health Spec. Bul. 57 (1931).

HYDROCEPHALUS IN FOXES*

By EARL F. GRAVES, *Poynette, Wis.*
Wisconsin State Fur Farm

Case 1: A young silver fox was presented for autopsy. There was no history. Autopsy showed very little brain tissue and a large amount of fluid present. The litter-mates of this animal

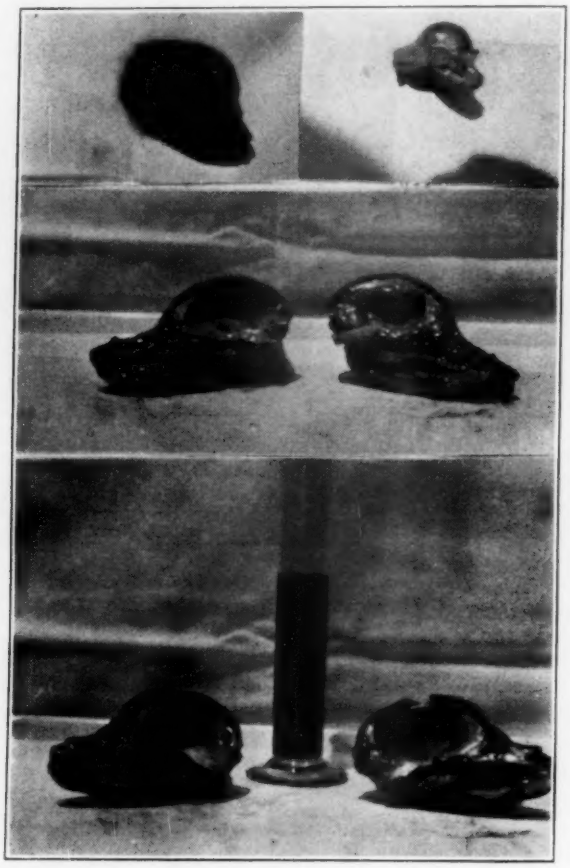

FIG. 1. Specimens from cases of hydrocephalus in foxes. Above, case 1; center, case 2; below, case 3.

were normal. The bones of the skull were almost paper-like in thickness and had many holes in them. This was an internal hydrocephalus case.

*Received for publication, July 6, 1937.

Case 2: A silver fox pup about nine weeks old was brought to the laboratory by its owner for observation. The history was meager; the owner said that the fox walked in circles. It was very stupid and would no longer eat or drink. The head appeared normal and the fontanels and cranial sutures were closed. My diagnosis was hydrocephalus and the animal was destroyed. Autopsy showed both ventricles to be so dilated as to be mere shells or membranes. There was slightly over 50 cc of fluid in the cavity. This would be a true case of internal hydrocephalus.

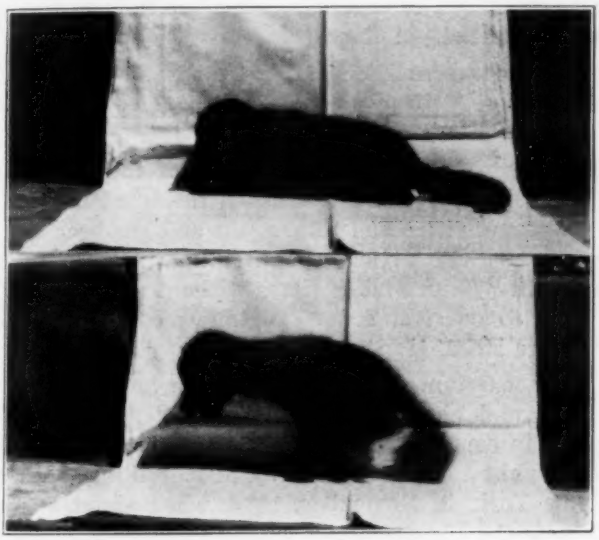

FIG. 2. Young blue fox with internal hydrocephalus.

Case 3: A young blue fox, born May 3, 1937, was a litter-mate to seven normal pups. Until it was about three weeks old, nothing unusual was noticed. It made normal growth and had held its own with the rest of the litter. However, it was found one morning in a state of exhaustion and near collapse. The animal was brought into the laboratory, warmed, and fed by hand. The head was enlarged slightly and the fontanel was open. The animal was unable to balance itself, held its head down, and appeared to be unable to focus the eyes.

After care by hand for a week, there was marked improvement. Although the fox was still unable to balance its body, it would eat unassisted and general body growth was visible. There was a very marked tendency for the animal to tip forward, as

may be seen in figure 2, and much effort was required by the hind quarters to remain on the ground. During the seventh week of age, there was so much general improvement that the animal was placed in a pen out of doors. During the eighth week, it suddenly began to walk backwards and at times in a circle.

For the last four days of its life, the fox would not eat or drink, either voluntarily or when material was placed in the mouth. The night before the fox was destroyed, it appeared to be very restless and at times "violently insane." Autopsy showed it to be a case of internal hydrocephalus. There was 53 cc of fluid present. The ventricles were but thin membranes and the fontanel had not yet closed.

Su-lin Makes the Front Page

Su-lin is less than a year old, but one of the most publicized animals in the world. She has crashed the front page of metropolitan newspapers time and time again and has been the subject of numerous feature articles published during recent months. She has had her picture taken times without number, and in this respect bids to outrival some of Hollywood's most glamorous motion-picture stars.

What has contributed to making Su-lin so popular with the press agents and photographers? Well, briefly, she is the only giant panda in captivity. She is a native of southwestern China and was brought to this country by Mrs. William H. Harkness, Jr., of New York, in November, 1936, when about a month old. Su-lin is now the big attraction at the Chicago Zoological Park, at Brookfield. It is reported that Mrs. Harkness declined an offer of $16,000 for Su-lin.

The Chicago climate is quite different from that of Su-lin's native habitat, which is high and dry. The warm, sultry days of June were not to Su-lin's liking, so plans are now under way to air-condition her quarters at Brookfield. Her meals are prepared by a trained nurse and a prominent Chicago veterinarian makes periodical visits to contribute to Su-lin's well-being.

There is no flock, however watched and tended,
  But one dead lamb is there;
There is no fireside, howsoe'er defended,
  But has one vacant chair.

—*Longfellow.*

INFLUENCE OF HOST FACTORS ON NEUROINVASIVENESS OF VESICULAR STOMATITIS VIRUS. I. Effect of age on the invasion of the brain by virus instilled in the nose. Albert B. Sabin and Peter K. Olitsky. Jour. Exp. Med., lxvi (1937), p. 15.

After intracerebral injection or nasal instillation of vesicular stomatitis virus in young or old mice, there was no evidence of a generalized systemic or blood infection. Within one hour after nasal instillation of as much as 100,000 m. c. l. d., no virus could be demonstrated in the nasal mucosa. Two days later, and thereafter, virus was abundant in the nasal mucosa of young mice, while among old mice it remained undemonstrable in some and present in small amounts in others. Virus was not detected in the anterior rhinencephalon of young and old mice within a few minutes and five hours after nasal instillation, but it was uniformly present on the second day. The essential differences in the further pathogenesis of the disease between the young mice which succumb with encephalomyelitis (5th day) and the old mice which survive without clinical signs of brain involvement is in the progression of the virus from the anterior rhinencephalon. Small minimal amounts of virus injected intracerebrally were shown to be disseminated quickly through the entire brain, killing old as well as young mice; virus so inoculated must spread differently from that which reaches the brain by the olfactory pathway.

INFLUENCE OF HOST FACTORS ON NEUROINVASIVENESS OF VESICULAR STOMATITIS VIRUS. II. Effect of age on the invasion of the peripheral and central nervous systems by virus injected into the leg muscles or the eye. Albert B. Sabin and Peter B. Olitsky. Jour. Exp. Med., lxvi (1937), p. 35.

Injection of vesicular stomatitis virus into the leg muscles of young mice gives rise to flaccid paralysis of the inoculated extremity as the first clinical sign of disease which is invariably fatal; old mice similarly injected survive without signs of illness. In young mice the virus multiplies at the site of inoculation and invades the sciatic nerve and spinal cord. In old mice intramus-

cular injection of 10 million m. c. l. d. leads to no local or systemic multiplication; thousands of m. c. l. d. of virus persist at the site of inoculation for at least four days. Injection of the virus directly into the sciatic nerve of old mice led to the typical paralytic disease in half the number of animals. The capacity of the virus to invade the central nervous system from the nerves but not from the muscle suggests the existence of a barrier in the muscle. Injection of the virus into the vitreous humor of the eye is followed by a fatal encephalitis in 15-day-old mice but one-year-old mice survive without signs of disease.

AN EFFECT OF GUINEA-PIG PASSAGE ON THE VIRUS OF RABIES. Weston E. Hurst. Brit. Jour. Exp. Path., xviii (1937), p. 205.

In both the rabbit and the guinea pig, rabies follows intravenous and intramuscular inoculation much more constantly and readily if the infecting material is guinea-pig brain rather than if it is rabbit brain. This property was not inherent in guinea-pig brain. The ratio between the minimal cerebral infecting dose and the minimal intravenous infecting dose is much lower with guinea-pig virus. The number of infective units of guinea-pig virus needed to infect by the intravenous route is much smaller than of rabbit virus. This effect of guinea-pig passage was observed only with viruses which were some passages removed from street virus. The Moroccan strain of virus behaved in a quite similar manner, though the results were highly irregular and difficult to read. No evidence was obtained of local multiplication of guinea-pig or rabbit virus at the site of intramuscular inoculation.

FOWL PEST: THE SUSEPTIBILITY OF MONKEYS, HEDGEHOGS AND OTHER ANIMALS. G. M. Findlay and R. D. MacKenzie. Brit. Jour. Exp. Path., xviii (1937), p. 258.

Rhesus monkeys are susceptible by intracerebral inoculation to certain strains of the virus of fowl pest. They react with fever and generally symptoms of ascending myelitis are present. Virus is present at death in the brain, liver and spleen and in association with the formed elements of the blood but not in the blood serum. Rhesus monkeys inoculated intraperitoneally or intranasally do not show any reaction, but subsequently develop immune bodies. Hedgehogs are susceptible to the virus of fowl pest when injected intracerebrally. Ducks are also susceptible on intracerebral but not on intramuscular injection. Pigeons may develop nervous symptoms after intraperitoneal and intracerebral

inoculation but die only when the virus is inoculated by the latter route.

PROPERTIES OF THE CAUSATIVE AGENT OF A CHICKEN TUMOR. XIII. Sedimentation of the tumor agent and separation from the associated inhibitor. Albert Claude. Jour. Exp. Med., lxvi (1937), p. 59.

The agent causing chicken tumor I can be separated from the other constituents of the tumor filtrate by means of high-speed centrifugation. Relative purification of the agent was obtained by means of differential centrifugation and washing in Tyrode's solution or in distilled water. The washed sediment gave opalescent solutions composed of minute particles of approximately but not exactly the same size. The tumor-producing activity of the washed sediment was significantly greater than that of the entire original filtrate. It is suggested that the gain in tumor-producing power was effected by the removal of an inhibiting factor known to occur normally in chicken tumor extracts.

LYMPH-NODES AS A SOURCE OF NEUTRALIZING PRINCIPLE FOR VACCINIA. Philip D. McMaster and John G. Kidd. Jour. Exp. Med., lxvi (1937), p. 73.

Regional lymph-nodes elaborate an antiviral principle when virus is brought to them by way of the lymphatics from the injected ear. This is demonstrable within four days after the virus inoculation. The antiviral principle was present in greater concentration in the extracts of lymph-nodes of the virus-injected side than in the undiluted blood serum procured at the same time. The possibility was excluded that the antiviral principle accumulated in the lymph-nodes from other parts of the body. The immunity following clinical vaccination may well be of lymph-node origin in great part.

PROPAGATION OF RABIES VIRUS IN TISSUE CULTURE. Leslie T. Webster and Anna D. Clow. Jour. Exp. Med., lxvi (1937), p. 125.

Rabies virus has been propagated in serum-Tyrode solution containing either embryo mouse brain or embryo chick brain. The culture virus reached a titre of 3×10^{-5} after incubation for four days at 37° C. and survived at least two months at 5° C. in the liquid or dry state. The culture virus remained active in the ice-box at 40° C. in fluid bulk or after freezing and drying. In

both cases the virus was infective for 30 days when diluted 10^{-1} and for 60 days when diluted 10^{-2}.

TURKEY-CHICKEN HYBRIDIZATION. J. P. Quinn, W. H. Burrows and T. C. Byerly. Jour. Hered., xxviii (1937), p. 169.

Fertile eggs can be obtained by artificially inseminating both turkey and chicken females with sperm of the other genus. Only a small percentage of the eggs, however, were fertile; attempts to increase fertility by various changes were unsuccessful. About 85 per cent of the embryos in fertile eggs died during the first day of incubation although one hybrid lived until fully developed and apparently was ready to hatch. This hybrid was intermediate in conformation between the chicken and turkey. Various external characteristics favored the chicken rather than the turkey.

THE MINIMUM VITAMIN-A AND CAROTENE REQUIREMENT OF CATTLE, SHEEP AND SWINE. H. R. Guilbert, R. F. Miller and E. H. Hughes. Jour. Nutri., xiii (1937), p. 543.

The minimum carotene requirement for all of the species studied was found to be 25 to 30 micrograms daily per kilogram of body weight, an amount in agreement with similar data on the rat. The minimum vitamin-A requirement on the basis of the analysis and criteria used was found to be 6 to 8 micrograms daily per kilogram body weight. Carotene was furnished by alfalfa and by crystalline carotene dissolved in cottonseed oil. Vitamin A was supplied by cod-liver oil. Excellent growth occurred at the above levels yet storage after extended periods was meager. Vitamin-A requirement is directly related to body weight rather than to energy requirement and the requirement of other species of mammals may be predicated on this basis. Indications of poor use of carotene by puppies is cited as a possible limitation in applying the generalization for carotene to carnivores. From the studies of storage at different levels of intake in animals, it appears that five to ten times the minimal intake is a desirable minimum for practical purposes.

THE INFLUENCE OF PARATHYROID HORMONE, UREA, SODIUM CHLORIDE, FAT AND OF INTESTINAL ACTIVITY UPON CALCIUM BALANCE. Joseph C. Curb, Dorothy M. Tibbets and Regina McLean. Jour. Nutri., xiii (1937), p. 573.

Parathyroid over-secretion has previously been shown to have no influence on fecal calcium excretion. The experiments re-

ported indicate that it has no consistent effect on intestinal absorption of calcium. An excellent organic solvent like urea does not influence calcium absorption from the intestine. Urea ingestion, however, does elevate the blood calcium level somewhat in exophthalmic goiter and hyerparathyroidism and increases urinary calcium excretion in all cases. This increased excretion is independent of diuresis. Calcium excretion in normal subjects is independent of voluntary constipation, cascara catharsis, or the ingestion of agar, large amounts of sodium chloride or of fat. Calcium excretion is therefore independent of many factors which might be expected to affect it and in health remains at a remarkably constant level considering the vast available store in the bones. That some factor greatly increases calcium absorption from the intestines is obvious from these experiments but its nature is still unknown.

TRANSMISSION OF FOWL LEUKOSIS. E. P. Johnson. Poultry Sci., xvi (1937), p. 255.

Evidence is submitted to show that mites (*Dermanyssus gallinae*) are capable of transmitting the leukosis agent from an affected bird to well birds under conditions that lead one to believe that this might take place under natural conditions. Five of 16 birds exposed to mites after feeding on a leukotic bird developed leukosis. The leukosis agent may be transferred mechanically from a leukotic bird to well birds by the common chicken-pox vaccination procedure. Seven of 38 birds developed one of the manifestations of leukosis following vaccination; the disease was presumably transferred mechanically by the brush used in the vaccination procedure. One control bird developed myeloid leukosis.

PARATYPHOID IN TURKEYS. V. A. Cherrington, E. M. Gildow and Pren Moore. Poultry Sci., xvi (1937), p. 226.

Paratyphoid infection in turkeys, and enzoötic septicemic disease of poults, has been observed in widely separated localities in Idaho. Poults died rapidly up to ten days of age; weakness and occasional diarrhea were the common symptoms. Older birds were more chronically affected and responded to hygienic practices. One-third to one-half of the hens in the breeding flocks that produced affected poults reacted when tested by the agglutination test with antigen made from a *Salmonella aertrycke* type organism. *S. aertrycke* type organisms were isolated from three out of 30 dead-in-the-shell poults cultured from one flock. The organism was not detected in 23 infertile eggs from the same

flock. The causative organism was isolated from the ovaries of two out of six hens examined. These birds were from a flock in which 13 reacted out of 24 birds tested. Attempts to eliminate the disease by fumigating the eggs in the incubator, by isolating the poults in sanitary quarters, and by the use of medication in the drinking water were not effective. Low vitality in the poults at hatching time and overheating in the brooder reduced resistance to infection. Clean 10-week-old poults brought in from outside sources and ranged with poults from the affected flock died extensively from paratyphoid infection.

FACTORS PRODUCING AND PREVENTING PEROSIS IN CHICKENS. V. G. Heller and Robert Penquite. Poultry Sci., xvi (1937), p. 243.

Experiments conducted over a period of five years, which consisted of careful external examinations of the legs, x-ray studies of the bones, and analysis of blood and bones demonstrated that the factors responsible for the cause and cure of rickets did not affect perosis. Excessive minerals usually aggravated the condition. A ration was developed that was effective in producing perosis in 76 to 100 per cent of the birds used. A drinking solution consisting of water extract of rice bran prevented the occurrence of defective legs. Water extracts of wheat bran, wheat gray shorts, wheat embryo and alfalfa did not prove very effective. The chickens consuming water extracts of rice bran had larger, smoother and better colored legs than the controls. The growth of the chickens was increased, disproving the statement that rapidly growing chickens are most susceptible. The ash of 70 pounds of rice bran added to 100 pounds of basal ration prevented perosis. A correlation exists between the manganese content and the curative properties of the ration, although it appears that other factors may be involved.

Use Partitions in Mixed Shipments of Live Stock

Much bruising and crippling of animals may be avoided by the use of strong partitions to separate different classes and kinds of live stock when shipped in the same truck. Surveys made at the Sioux City, Iowa, market by Harry J. Boyts, Live Stock Commissioner, have shown that there are twice as many bruises on hogs hauled with cattle without partitions, as are found in straight loads of hogs shipped by truck.

Regular Army

Captain Harry R. Leighton is relieved from further assignment and duty at the Seattle Quartermaster Depot, Seattle, Wash., effective in time to comply with this order, is then assigned to station at Fort Williams, Maine, and will proceed at the proper time to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about September 22, 1937, for New York, N. Y.; upon arrival in New York will proceed to Fort Williams, Maine, and report to the commanding officer for duty.

The promotion of each of the following-named officers in the Veterinary Corps, to the grade of lieutenant colonel, is announced:

Major Kenneth E. Buffin, with rank from June 11, 1937.

Major William R. Wolfe, with rank from June 23, 1937.

Veterinary Reserve Corps

NEW ACCEPTANCES

Barchfeld, Wm. Powell.....1st Lt...4332 Luster St., Pittsburgh, Pa.
Black, Joseph Marion.....1st Lt...306 W. Houston, Marshall, Texas.
Brundrett, Frank Wilfred...1st Lt...311 N. Windomere, Dallas, Texas.
Collins, Horace Reynolds, Jr. 1st Lt...1814 Anderson Ave., Manhattan, Kans.
Craige, John Etherington....1st Lt...1317 So. 57th St., Philadelphia, Pa.
Crawford, Edwin Morris....1st Lt...Box 542, Kans. State Col. Manhattan, Kan.
Deal, Blakey Thatcher.....1st Lt...5350 Webster St., Philadelphia, Pa.
Eagelman, Jas. Garfield, Jr..1st Lt...Geigertown, Berks County, Pa.
Fechner, Walter Wallace....1st Lt...R. R. No. 1, Alta Vista, Kan.
Flack, George Russell.....1st Lt...2825 W. Somerset St., Philadelphia, Pa.
Harkins, Woodrow Wilson..1st Lt...Alpine, Ala.
Horn, Wiley Henry.....1st Lt...1953 Prairie, Forth Worth, Texas.
Martinez, Homer Thomas...1st Lt...Hebbronville, Texas.
Preston, Edward Charles....1st Lt...613 Rising Sun Ave., Philadelphia, Pa.
Richter, Carl Morris.....1st Lt...Elizabethville, Pa.
Sadow, Irving Jay.....1st Lt...2922 W. 24th St., Brooklyn, N. Y.
Saturen, Israel Moshe.....1st Lt...309 N. Marshall St., Philadelphia, Pa.
Schott, Francis Joseph.....1st Lt...Nacogdoches, Texas.
Spong, Lawrence Eric.....1st Lt...Enterprise, Kan.
Stadler, Robert John .....1st Lt...143 Kelsey St., New Britain, Conn.
Wainright, Charles Roy.....1st Lt...R. R. No. 1, Box 101A, Tulsa, Okla.
Watson, Douglas Fleming...1st Lt...Stanwood, Mount Kisco, N. Y.
Watson, James Howard.....1st Lt...Shawnee, Kan.

Weaver, Wayne Clinton.....1st Lt...Andreas, Pa.
Willard, Horace Randolph..1st Lt...Giddings, Texas.

PROMOTIONS

To

Durigg, John Raymond.....1st Lt...East Chestnut St., Oxford, Ohio.
Odom, Houston .....Capt...Auburn, Ala.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Flack, Geo. Russell.....1st Lt...Hdqrs. 3rd Corps Area, Baltimore, Md.
Bechtol, Lauren L.....1st Lt...Fort Knox Dist., Fort Knox, Ky.
Bate, Louis B. .....1st Lt...Boise, Idaho.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Fisherman, Henry .....1st Lt...Silver City Dist., Silver City, N. Mex.
Leenerts, Theodore H.....1st Lt...Pocatello, Idaho.
Thompson, Thomas M.....1st Lt...Presidio of San Francisco, Calif.

New Laboratory for Alabama

On a site of 40 acres, deeded by the Alabama Polytechnic Institute, at Auburn, Ala., the U. S. Department of Agriculture will establish a regional laboratory devoted to research on animal diseases. This laboratory, for which plans have just been approved, is one of a series of research laboratories established in major agricultural regions as authorized by the Bankhead-Jones Act. Construction work will begin at once and officials of the U. S. Bureau of Animal Industry expect to have the laboratory in operation during this fiscal year.

The laboratory will deal primarily with major problems affecting the health of domestic animals and poultry in 13 southern states: Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississipi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas and Virginia.

In order to be of the greatest service to the region, the work of the new laboratory will be planned and conducted in coöperation with the agricultural experiment stations of the states named. Projects already formulated deal with a more effective control of Johne's disease of cattle and coccidiosis of live stock, including poultry. Projects involving diseases caused by bacteria and viruses will be under the general supervision of Dr. H. W. Schoening, Chief of the Pathological Division, U. S. Bureau of Animal Industry. Those involving diseases caused by parasites will be supervised by Dr. Benjamin Schwartz, Chief of the Zoölogical Division of the same Bureau.

Veterinarian Appointed to Milk Sanitation Advisory Board

Dr. J. G. Hardenbergh, of Plainsboro, N. J., has been appointed to membership on the Public Health Service Milk Sanitation Advisory Board. This appointment is distinctly a

DR. J. G. HARDENBERGH

recognition of the veterinary profession, as previous to the appointment of Dr. Hardenbergh there had been no veterinarian on the Board, which was organized so that the United States Public Health Service might have at its command the technical advice of a comprehensive group of experts in the various phases of the public health control of milk supplies, and in

allied problems relating to the production, processing and distribution of milk.

Other members of the Board are:

Mr. H. A. Whittaker, Director, Division of Sanitation, Minnesota State Health Department, Minneapolis, Minnesota.

Mr. C. A. Abele, Director, Bureau of Inspection, State Health Department, Montgomery, Ala.

Dr. Paul B. Brooks, Deputy Commissioner of Health, State Health Department, Albany, N. Y.

Mr. V. M. Ehler, Director, Bureau of Sanitary Engineering, State Board of Health, Austin, Texas.

Mr. H. A. Kroeze, Director, Bureau of Sanitary Engineering, State Board of Health, Jackson, Miss.

Mr. E. S. Tisdale, Chief Engineer, Division of Sanitary Engineering, State Health Department, Charleston, W. Va.

Mr. Ernest Kelly, Chief, Division of Market-Milk Investigations, U. S. Department of Agriculture, Washington, D. C.

Dr. D. B. Peck, Bowman Dairy Company, Chicago, Ill.

Mr. Loomis Burrell, Cherry-Burrell Corporation, Little Falls, N. Y.

Mr. Seth W. Shoemaker, Scranton, Pa.

Mr. Leslie C. Frank, Sanitary Engineer in Charge, Office of Milk Investigations, U. S. Public Health Service, Washington, D. C.

Mr. Whittaker is chairman of the Board, and Mr. Frank is secretary. Dr. Peck represents the International Association of Milk Dealers, Mr. Burrell the Dairy and Ice Cream Machinery and Supplies Association, and Mr. Shoemaker the Certified Milk Producers Association of America, but the participation of these members in the deliberations of the Board does not necessarily imply approval of the recommendations of the Board by the organizations represented.

Dr. Hardenbergh is Laboratory Director for the Walker-Gordon Laboratory Company at Plainsboro, N. J. secretary of the Veterinary Medical Association of New Jersey, and chairman of the A. V. M. A. Special Committee on Food Hygiene.

Doctor Foster Writes on the Horse

Dr. J. P. Foster, of Minneapolis, Minn., has written some more very interesting articles that have appeared in *The Thoroughbred Record* during recent months. The articles on "Roaring of Horses," "Periodic Ophthalmia," "Azoturia" and "Navel Ill" contained lots of information of interest to owners of horses. In each article, however, the author pointed out the desirability of consulting veterinarians in such conditions. Dr. Foster also contributed an interesting story entitled, "A Tale of Two Horsemen," and in another article, "An Obstetrical Problem," he discussed superfecundation and superfetation. Another article in the series covered "Sex Determination."

OKLAHOMA VETERINARY MEDICAL ASSOCIATION

The twenty-second semi-annual meeting of the Oklahoma Veterinary Medical Association was held at the Skirvin Hotel, Oklahoma City, June 7-8, 1937, with an attendance of over 100 veterinarians. The meeting was called to order by Dr. W. L. Christy, of Tonkawa. Following the invocation, Mr. F. G. Baker delivered the address of welcome and the response was given by Mr. Joe C. Scott, president of the Oklahoma State Board of Agriculture. The following literary program was then presented:

"Undulant Fever," by Dr. Louis Dakil, Oklahoma City General Hospital, Oklahoma City.

"The Dependability of the Agglutination Test for Bang's Disease," by Dr. Walter Wisnicky, Director of Live Stock Sanitation, Madison, Wis.

"Gastro-Intestinal Infections of Dogs and Their Differentiation." "Canine Infectious Jaundice," and "Canine Distemper, the Disease and Its Treatment," by Dr. Norman J. Pyle, Pearl River, N. Y.

"The Relation of the Veterinarian to Safe and Sane Milk Inspection," Dr. Walter H. Martin, El Reno.

"Dobbin's Return Calls for a Discussion of Equine Dentistry," by Dr. E. H. Leonhard, Oklahoma City.

"Swine Erysipelas," by Dr. Roy T. Fisher, Stillwater.

"The Veterinarian's Place in the Community," by Dr. S. E. Douglas, Tahlequah.

"Veterinary Service of the C.C.C.," by Capt. John H. Wirtz, Oklahoma City.

Discussions of the various papers and addresses were conducted at the close of each session by Dr. A. T. Kinsley, of Kansas City, Mo., Dr. W. F. Irwin and Dr. O. E. Robinson.

A banquet was served the evening of the first day to 131 persons. Dr. Kinsley acted as toastmaster.

A resolution was adopted asking that a member of the veterinary profession be included, along with physicians, nurses and members of other professions, in public health units, under the Social Security Act. Favorable action was taken regarding a series of radio broadcasts, to be put on by the Association, in agreement with a recommendation of the A. V. M. A. Committee

on Public Relations. The Public Relations Committee of the O. V. M. A. was instructed to work out the details.

The entertainment of the ladies was in charge of the Ladies' Auxiliary of the O. V. M. A.

F. Y. S. Moore, *Sec.-Treas.*

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The fourteenth semi-annual meeting of the State Veterinary Medical Association of Texas was held at the Texas A. and M. College, College Station, June 8-9, 1937. The attendance was one of the largest in the history of its 27 years of existence. There were 175 present. The program which follows proved to be very interesting and the papers were freely discussed:

"The Relation of Maternal Vitamin-A Deficiency to Microphthalmia in Pigs," by Fred Hale, College Station.

"Piroplasmosis of Dogs," by Dr. R. C. Dunn, College Station. A discussion of the symptoms, causative organism, technic of taking blood samples, diagnosis and treatment.

"Gastro-Intestinal Infections of Dogs," by Dr. Norman J. Pyle, Pearl River, N. Y.

"Demonstrations of Some Poisonous Plants," by Drs. P. W. Burns and G. T. Edds, College Station.

The clinic for small animals was in charge of Dr. E. R. Frank, Kansas State College, Manhattan. Dr. A. A. Lenert, College Station, discussed "Infectious Granuloma of Dogs" and demonstrated the surgical removal. Dr. R. A. Self, Dallas, discussed the trimming of terriers' ears and gave a demonstration. Dr. L. E. Casey, Dallas, performed an operation for correcting faulty tails (squirrel tails) in terriers.

Other subjects presented were:

"Bang's Disease Eradication Program in Texas," by Dr. H. L. Darby, U. S. B. A. I., Fort Worth.

"Problems of Bang's Disease Eradication," by Dr. T. O. Booth, Fort Worth.

"Trichomonad Abortion in Cattle," by Dr. H. Schmidt, College Station.

"Important Parasites Affecting Live Stock in Texas," by Dr. Benjamin Schwartz, U. S. B. A. I., Washington, D. C.

The clinic for large animals was in charge of Dr. E. R. Frank, who was also the principal speaker.

The student chapter of the American Veterinary Medical Association at the School of Veterinary Medicine, Texas A. and M. College, proposed the erection of a suitable memorial to the

former dean, Dr. Mark Francis. By unanimous vote, sponsorship of the project was given to the proponents.

M. B. STARNES, *Corresponding Secretary.*

WYOMING VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Wyoming Veterinary Medical Association was held in Sheridan, June 21-22, 1937.

A successful program, consisting of papers and a clinic, was held. Among the veterinarians contributing to the program were: Drs. W. J. Butler and G. W. Cronen, State Veterinarian and Inspector-in-Charge, U. S. Bureau of Animal Industry, respectively, of Helena, Mont.; Dr. Frank Breed, Lincoln, Neb.; Dr. H. J. Shore, Fort Dodge, Iowa, and Dr. James Farquharson, Fort Collins, Colo. The latter demonstrated a number of surgical operations.

The Association voted an appropriation of $20.00 to the A. V. M. A. Exhibit Fund being raised for financing educational exhibits.

The following officers were elected for the ensuing year: President, Dr. L. H. Scriver, Laramie; vice-president, Dr. W. H. Lee, Powell, and secretary-treasurer, Dr. H. D. Port (reëlected), Cheyenne.

H. D. PORT, *Secretary-Treasurer.*

CALIFORNIA STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the California State Veterinary Medical Association was held in Long Beach, June 21, 22 and 23, 1937. The facilities provided by the local committee and the Long Beach Municipal Convention Bureau were excellent.

The program was featured by a discussion of "Diseases of the Eye," by George L. Kilgore, M. D., and an interesting illustrated lecture by C. E. ZoBell on "Some Practical Aspects of the Microbiology of the Sea." In addition there were the usual papers of professional interest and the business sessions. The business sessions approved coöperation with the American Veterinary Medical Association Committee on Public Relations.

A new veterinary practice act passed by the legislature was explained by Dr. J. M. Arburua, of San Francisco. It was de-

cided to make a definite attempt to discourage the practice of allowing lay assistants to do veterinary work in the hospital or in the field.

The Ladies' Auxiliary provided appropriate entertainment for the ladies who attended the meeting.

The following officers were elected for the ensuing year: President, Dr. Geo. M. Simmons, San Francisco; 1st vice-president, Dr. E. E. Jones, Los Angeles; 2nd vice-president, Dr. R. A. Ball, Modesto; 3rd vice-president, Dr. L. F. Conti, San Diego; treasurer, Dr. W. E. Phelps, Redlands, and secretary, Dr. Chas. J. Parshall (reëlected), Brentwood. Drs. John L. Tyler, of Whittier, and G. N. Miller, of Covina, were elected delegate and alternate, respectively, to the A. V. M. A. House of Representatives.

CHAS. J. PARSHALL, *Secretary.*

MISSOURI VETERINARY MEDICAL ASSOCIATION

The forty-sixth annual meeting of the Missouri Veterinary Medical Association was held in Marshall, June 24-25, 1937, with a registration of almost 150 veterinarians. Dr. H. A. Wilson, of Malta Bend, presided at the meeting and gave an address.

The following program was given:

"Hog Cholera and Associated Diseases," by Dr. G. E. Whipple, Omaha, Neb.

"Some Thoughts on Nutrition," by Dr. Kent R. Dudley, Iola, Kan.

"Calcium, Phosphorus and Magnesium Deficiencies" (illustrated), by Dr. O. F. Reihart, Omaha, Neb.

"Meat Inspection," by Col. Robert J. Foster, V. C., U. S. Army, Washington, D. C.

"Recent Developments in the Diagnosis of Canine Distemper and Allied Diseases," by Dr. H. C. Smith, Fort Dodge, Iowa.

"Advancement in the Diagnosis and Treatment of Poultry Diseases," by Dr. A. J. Durant, Columbia, Mo.

"Observations of Practical Significance in Small-Animal Hospital Management," by Dr. H. W. Young, Kansas City, Mo.

"Vitamins and Their Importance in Animal Health," by Mr. Frank J. Holt, Chicago, Ill.

The banquet on the evening of the first day of the meeting was presided over by President H. A. Wilson. Col. Robert J. Foster, as president of the A. V. M. A., gave a talk on "The American Veterinary Medical Association, Your Organization." He so stimulated interest in the national association that several of those present filed applications for membership. Mr. Tom Collins, radio and newspaper humorist, enlivened the occasion with his subject, "Mirth and Humor."

The following officers were elected for the ensuing year: President, Dr. T. E. Wilke, West Plains; vice-president, Dr. W. H. Bailey, Saint Joseph; Third District trustee, Dr. S. W. Haigler, Saint Louis, and secretary-treasurer, Dr. C. L. Campbell (re-elected), Kansas City.

C. L. CAMPBELL, *Secretary-Treasurer.*

NORTH DAKOTA VETERINARY ASSOCIATION

The thirty-third annual meeting of the North Dakota Veterinary Association was held at the Agricultural College, Fargo, June 24-25, 1937. There was an attendance of about 40 North Dakota and Minnesota veterinarians. The following program was of unusual interest and had an enthusiastic reception, so that the meeting was indeed a successful one:

"Observations on Sheep Scab," by Dr. B. K. Bjornson, Fargo.
"Sheep Feeding Problems and Sheep Diseases," by Dr. L. D. Frederick, Chicago, Ill.
"Malignant Catarrhal Fever of Cattle," by Dr. T. O. Brandenburg, Bismarck.
"Nutritional Requirements and Deficiencies of Swine" and "Blackleg and Its Complications," by Dr. Frank Breed, Lincoln, Neb.
"Some Problems in Equine Surgery," by Dr. Geo. R. Fowler, Ames, Iowa.
"Digestive Disturbances in Cattle," by Dr. F. E. Walsh, Ames, Iowa.

A combined dinner and social hour, followed by the concluding business session, was held during the evening of the first day. The afternoon of the second day was devoted to a clinic and demonstration program, by Drs. Fowler and Walsh, which were arranged by the Clinic Committee.

Officers for 1938 were elected as follows: President, Dr. Walter Fleenor, Fairmount; vice-president, Dr. J. W. Robinson, Garrison, and secretary-treasurer, Dr. M. C. Hawn, Fargo.

LEE M. RODERICK, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The fifty-third semi-annual meeting of the Veterinary Medical Association of New Jersey was held on the afternoon of July 8, 1937, in conjunction with the Eastern States Veterinary conference, at the Hotel New Yorker, New York City.

The meeting of the Association was a business session only, the literary and clinical program being furnished by the Conference. This meeting must be recorded as one of the best; from beginning to end, the papers, scientific reports and clinical demonstrations were of a very high degree of excellence.

At the business session of our State Association. Dr. R. A. Hendershott, of Trenton, and Dr. J. G. Hardenbergh, of Plainsboro, were designated as delegate and alternate, respectively, to the A. V. M. A. House of Representatives meeting in Omaha in August. The Association also voted to appropriate $75.00 toward defraying the expenses of the delegate.

Two amendments to the Constitution and By-Laws presented at the meeting in January were adopted. The first provides for membership of veterinarians in federal employ without payment of dues and reads as follows:

> A veterinarian in the employ of the federal government, who is stationed in New Jersey and who is a member in good standing of a state veterinary association and of the American Veterinary Medical Association, may be admitted to active membership in this Association without the payment of the initiation fee, but upon payment of the annual dues, provided, that application for membership shall have been made in the regular form, passed upon by the Executive Committee and the applicant elected by majority vote as in the preceding paragraph. If the applicant is not already a member of another state veterinary association, then he shall pay the initiation fee as provided in the first paragraph of this article.

The second amendment relates to the dropping, automatically, of members delinquent in dues three years, without formal action of the Association and also to the reinstatement of such delinquent members.

J. G. HARDENBERGH, *Secretary.*

WASHINGTON STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Washington State Veterinary Medical Association was held at the Multnomah Hotel, Portland, Ore., July 12, 1937, during the meeting of the Northwest Veterinary Medical Association. Dr. S. S. Worley, of Bellingham, presided while the following business was transacted.

Henceforth, the annual business meeting of the Association will be held in the state of Washington, the date and place to be selected by the Executive Committee. This meeting will be held in the fall of the year and in mid-state.

The fiscal year of the Association was changed to the calendar year. All incumbent officers will hold office until the October meeting as provided.

Veterinarians in the employ of the U. S. Bureau of Animal Industry, the U. S. Army and other federal services, who are members of the American Veterinary Medical Association, will be admitted to membership in the Washington Association on payment of the anual dues without initiation fee.

The records, accounts and minutes of the Association, since its organization in 1909, have been transcribed in duplicate and bound in book form. One copy, with the originals, is to be placed in the library of the State College of Washington and the other copy will be retained in the files of the Secretary.

At the request of the A. V. M. A. Committee on Public Relations, the president was authorized to appoint a similar committee for the Washington Association to function with the Committee on Public Relations of the national organization. The secretary is to be a member of the committee.

V. C. PAUHLMAN, *Secretary-Treasurer.*

NORTH CAROLINA AND VIRGINIA STATE VETERINARY MEDICAL ASSOCIATIONS

The thirty-sixth annual meeting of the North Carolina State Veterinary Medical Association was a joint meeting with the forty-fourth annual meeting of the Virginia State Veterinary Medical Association, held at Wrightsville Beach, N. C., July 15-16, 1937. Approximately 75 veterinarians were in attendance from the two states and a number of guests and visitors from other states.

President B. J. Lindley, of the North Carolina Association, presided on the first day and the following program was given:

"Mastitis," by Dr. Ashe Lockhart, Kansas City, Co.

"Some Practical Aspects of Surgical Technic," by Dr. W. F. Guard, Ohio State University, Columbus, Ohio.

"Animal Mortality and Morbidity Statistics," by Mr. Frank Parker, Statistician, U. S. Department of Agriculture, Raleigh, N. C.

A small-animal clinic was conducted by Dr. E. B. Dibbell, of Baltimore, Md., in the afternoon and a number of cases of skin diseases were presented and discussed.

Dr. I. D. Wilson, Virginia Polytechnic Institute, Blacksburg, Va., was toastmaster at the evening banquet. He introduced the distinguished guests and then presented Hon. John J. Burney, of Wilmington, Solicitor of the Eighth North Carolina District, who made a humorous address.

On the second day Dr. I. P. Gilbert, president of the Virginia Association, presided. The following program was given:

"Skin Diseases of Dogs," by Dr. E. B. Dibbell, Baltimore, Md.

"Public Interest in Control of Animal Diseases," by Mr. F. H. Jeter, Agricultural Editor, State College, Raleigh, N. C.

"Swine Erysipelas," by Dr. H. W. Schoening, U. S. Bureau of Animal Industry, Washington, D. C.

"Animal Health Insurance," by Dr. J. J. Jones, La Plata, Md.

"Lameness of the Horse" (a moving picture), by Dr. W. F. Guard, Columbus, Ohio.

At the business meeting of the North Carolina Association an amendment to the By-laws was adopted providing that "any graduate veterinarian who can produce a certificate of good standing in another state veterinary medical association may be admitted to membership upon the payment of $3.00, which would be the dues for the current year."

The following officers of the North Carolina Association were elected for the ensuing year: President, Dr. G. A. Ferguson, Reidsville; 1st vice-president, Dr. G. C. Monroe, Greensboro; 2nd vice-president, Dr. D. C. Beard, Sanford; secretary-treasurer, Dr. P. C. McLain, Charlotte, and directors, Dr. H. J. Rollins, Rockingham and Dr. M. M. Leonard, Asheville.

J. H. BROWN, *Secretary.*

VIRGINIA STATE VETERINARY MEDICAL ASSOCIATION

The forty-fourth annual meeting of the Virginia State Veterinary Medical Association was a joint meeting with the North Carolina State Veterinary Medical Association, held July 15-16, 1937, at Wrightsville Beach, N. C. (For details concerning the literary program, see foregoing report.)

At the business session of the Virginia Association, the following officers were elected for the ensuing year: President, Dr. O. F. Foley, Harrisonburg; 1st vice-president, Dr. J. E. Greer, Pulaski; 2nd vice-president, Dr. R. Todd Gregory, Fredericksburg; secretary, Dr. A. J. Sipos (reëlected), Richmond, and treasurer, Dr. R. E. Brookbank (reëlected), also of Richmond.

Three applicants were admitted to membership. The sum of $25.00 was appropriated toward the veterinary exhibits which are to be placed in the New York World's Fair and the San Francisco Exposition in 1939 under the auspices of the national Association.

A. J. Sipos, *Secretary.*

EASTERN STATES VETERINARY CONFERENCE

The first Eastern States Veterinary Conference was held at the Hotel New Yorker, New York, N. Y., July 7-8-9, 1937. Dr. C. P. Zepp, of New York City, was chairman of the Organizing Committee and chairman of the Small-Animal Section. Dr. Cassius Way was chairman of the Large-Animal Section.

Veterinary organizations from the following states participated in the conference: Connecticut, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont and the District of Columbia. More than 500 veterinarians were registered. The literary program was well diversified and the several sessions were well attended. Prominent veterinarians from eleven states took part in the program.

The large-animal clinic drew much attention because it was the first one to be held in a New York City hotel. A clinic including horses and cattle in the heart of a great metropolis was so unusual that it attracted newspaper writers and news-reel cameramen to the meeting to obtain stories and pictures of such unusual proceedings as casting a horse on a ballroom floor, taking x-ray pictures of a horse's leg, and putting a horse under general anesthesia.

No attempt is being made here to cover the thirty or more addresses and papers which were given, as arrangements have been made for the publication of the proceedings, a copy of which will be furnished every veterinarian who attended.

E. C. W. S.

Texas Breeders Organize

Because of the decline in the number of horse and mule colts in Texas, an organization for stimulating interest in breeding was formed recently. It will be known as the Texas Horse, Jack and Mule Breeders' Association, according to a report in *The Cattleman*. Many breeders in the Lone Star State have found it necessary to raise their own jacks, as there is no supply available for importation.

First Airboy: "Ha! Ha! I flew into a flock this morning."
Second Airboy: "Scare you much Ed?"
First Airboy: "Naw, but I was covered with goose flesh."

—*Successful Farming.*

NECROLOGY

LONNIE P. ARNOTT

Dr. Lonnie P. Arnott, of Lawrence, Kan., died on January 27, 1937. Death was due to secondary anemia complicated by an obstruction of the stomach.

Born in West Virginia, in 1869, Dr. Arnott moved to Kansas in 1894. He was graduated from the Kansas City Veterinary College in 1906 and entered practice at McPherson, Kan. Later he was located at Wakeeney, same state, and in 1920 he went to Lawrence, where he remained until his death. His survivors include one daughter, two sons, one sister and four brothers.

ROBERT LOWELL MASON

Dr. Robert L. Mason, of Trenton, N. J., died at Mercer Hospital, Trenton, April 12, 1937, at the age of 45. He was a native of Lawrence, Kan., and received his veterinary degree from the University of Pennsylvania in 1917. Later he studied human medicine at the Howard Medical School, in Washington, D. C. Ever since his graduation in 1932, he had been located in Trenton. He is survived by his widow, one son, his father, one brother and one sister.

WILLIAM N. HOBBS

Dr. William N. Hobbs, of Kensington, Kan., died at the home of his daughter, in Clyde, Kan., on May 3, 1937, the victim of a paralytic stroke. He was in his 76th year. Dr. Hobbs was a graduate of the Kansas City Veterinary College, class of 1896, and had been engaged in general practice ever since his graduation. He located first at Holton, Kan., and disposed of his practice there about 1908, to accept a position on the faculty of the Saint Joseph Veterinary College. He located in Kensington in 1913. He is survived by his widow, one son and two daughters.

JOHN H. DEDRICK

Dr. John H. Dedrick, of Albany, Ind., died at his home, June 27, 1937. He had been in poor health for the past two years. He was a graduate of the New York College of Veterinary Surgeons, class of 1896, but had retired from active practice a number of years ago. He was formerly located in Muncie, Ind. The survivors are the widow, a brother and a niece.

JAMES F. WILLIAMS

Dr. J. F. Williams, of Highland Center, Iowa, died at his home on February 1, 1937, following a long illness. He was 61 years old. He was a graduate of the Kansas City Veterinary College, class of 1911, and had practiced at Highland Center for quite a number of years.

JOHN L. HICKMAN

Dr. John L. Hickman, of Piedmont, Kan., died in the Barnes Hospital, Saint Louis, Mo., on March 2, 1937, at the age of 65 years. He had been critically ill for several weeks. He was a graduate of the Kansas City Veterinary College, class of 1909, and had practiced at Piedmont for a number of years. He is survived by his widow, one son and two daughters.

JAMES M. MECRAY

Dr. James M. Mecray died at the home of his daughter in New Hope, Pa., on March 18, 1937, after an illness dating back several years. He was a graduate of the University of Pennsylvania, class of 1895, and practiced for many years at Maple Shade, N. J. He is survived by a daughter and a sister.

ANDREW JOSEPH SCHAUF

Dr. Andrew J. Schauf, of Richland Center, Wis., died on January 1, 1937. He was born in Germany, March 28, 1867, and was a graduate of the Kansas City Veterinary College, class of 1911. He had been in general practice at Richland Center ever since his graduation. Three sisters and two brothers survive the deceased.

W. B. McALESTER

Dr. W. B. McAlester, of South McAlester, Okla., died on April 20, 1937, following a long illness. He was state representative from Pittsburg County, and a son of the late J. J. McAlester, founder of the city of that name. Dr. McAlester was a graduate of the Kansas City Veterinary College, class of 1904, but had not been actively identified with his profession for sometime. He was an extensive landowner, a raiser of live stock and interested in politics. He is survived by his widow and two children.

WILLIAM L. FOWLER

Dr. William L. Fowler, of Greenwich, Conn., was found dead of carbon monoxide poisoning beside his automobile on a street near his home, June 9, 1937. He was 70 years of age and a graduate of the New York College of Veterinary Surgeons, class of 1898. His entire professional career had been spent in Greenwich.

G. E. C.

SAMUEL C. ROSS

Dr. Samuel C. Ross, of Plainview, Texas, was killed in an automobile accident, near San Angelo, Texas, on June 29, 1937.

Born in DeLeon, Texas, in 1881, he studied veterinary medicine at the Kansas City Veterinary College. He was graduated in 1915 and had practiced most of the time since at Plainview. For several years he had been City Meat, Milk and Sanitary Inspector.

Dr. Ross joined the A. V. M. A. in 1921. He is survived by two sons.

S. R.

OWEN J. McGURTY

Dr. Owen J. McGurty, of Paris, Ill., died in Saint Anthony's Hospital, in Terre Haute, Ind., on July 2, 1937, after an illness of several years.

Born at Charleston, Ill., March 14, 1867, Dr. McGurty entered the Chicago Veterinary College after receiving an academic education. He practiced at Paris for many years and conducted a drug store there a part of the time.

Dr. McGurty joined the A. V. M. A. in 1920. He is survived by a daughter and a sister.

LEWIS NELSON JARGO

Dr. Lewis N. Jargo, of Mason City, Iowa, died in his home, July 3, 1937. He had been in poor health for about three years and had been bedfast for almost six months.

Born in Cottage Grove, Wis., September 22, 1876, Dr. Jargo attended high school and then entered the Ontario Veterinary College. He graduated in 1900 and some years later resumed his veterinary studies at the McKillip Veterinary College. He received a degree from this institution in 1908. He had been a veterinary inspector in the U. S. Bureau of Animal Industry for 29 years. He entered the meat inspection service on July 1, 1908, at Chicago, and served at various stations before going to Mason City.

Dr. Jargo joined the A. V. M. A. in 1918. He is survived by two brothers and three sisters. A large number of veterinarians attended the funeral on July 6, six prominent members of the profession acting as pall bearers.

CARL CURTIS HARTING

Dr. Carl C. Harting, of Ladoga, Ind., died in Culver Hospital, Crawfordsville, Ind., July 9, 1937, after an illness of almost three weeks.

Born near Jamestown, Ind., June 13, 1886, Dr. Harting was graduated from the Indiana Veterinary College in 1911. He practiced at Crawfordsville for 18 years, then in Bainbridge, later at Waveland, and located at Ladoga three years ago. He is survived by his widow, one daughter, two step-sons and three brothers.

WILLIAM ALBERT THOMAS

Dr. William A. Thomas passed away on July 6, 1937, at the age of 86 years, at his home in Lincoln, Neb.

Born in Willamantic, Conn., Dr. Thomas came to Lincoln shortly after his graduation from Iowa State College in 1880. He was one of the earliest graduate veterinarians in Nebraska, if not the first one. He served as State Veterinarian for a time during these early days. After retiring from practice, several years ago, he devoted his time to the development of his small farm near the city, but retained a lively interest in the profession and its advances. His was a lovable character, endearing

to his many devoted friends. Always an entertaining visitor, he liked to discourse on the status of the veterinary profession of the early days, and on his experiences with dourine, Texas fever, anthrax, and other disorders.

Dr. Thomas is survived by three daughters, thirteen grandchildren, a brother and a sister.

L. V. E.

JAMES M. MILLER

Dr. James M. Miller, of Benton Harbor, Mich., was killed in an automobile accident on U. S. 12, at the southern city limits of St. Joseph, Mich., on July 13, 1937. He was returning from a professional call.

Born December 9, 1878, at Bay City, Mich., Dr. Miller had been a resident of Benton Harbor for 48 years. He was a graduate of the old Benton Harbor College and the McKillip Veterinary College (1913). During the World War, Dr. Miller was commissioned as a second lieutenant in the Veterinary Corps and was at Camp Greenleaf, Ga., for about three months.

Dr. Miller joined the A. V. M. A. in 1929. He was widely known throughout Berrien County and was an unsuccessful candidate for sheriff on the Democratic ticket in 1932 and again in 1936. He is survived by his widow (née Elsie Rohnoser), his parents, two brothers and one sister.

EDDIE VICTOR ALEXANDER

Dr. E. V. Alexander, of Indianapolis, Ind., died at his home on July 14, 1937, following an illness of five weeks.

Born in Greencastle, Ind., January 30, 1881, Dr. Alexander had lived in Indianapolis nearly all his life. He was a graduate of the Indiana Veterinary College, class of 1907, and had spent his entire professional career in the service of the U. S. Bureau of Animal Industry. As a tenor vocalist he was active in musical circles in Indianapolis. He was a member of the Mystic Tie Quartet and the Scottish Rite Choir.

Dr. Alexander joined the A. V. M. A. in 1916. He was a member of the National Association of B. A. I. Veterinarians. He was a charter member of the Northwood Christian Church and a member of Center Lodge, F. and A. M., and the Scottish Rite. He is survived by his widow (neé Bessie Clodfelter), three daughters, four sisters and two brothers.

T. A. S.

ESMOND VERLE SMITH

Dr. Esmond V. Smith, of Canyon City, Ore., was drowned in Canyon Creek, on May 2, 1937. According to newspaper reports, he was driving a car which left the road and overturned in the creek. His body was not recovered until several days later. Other occupants survived.

Born at Lost Springs, Kan., June 12, 1912, Dr. Smith attended the Puyallup (Wash.) High School and later entered the State College of Washington for the study of veterinary medicine. Following his graduation in 1935, he entered the employ of the U. S. Bureau of Animal Industry and was engaged in Bang's disease control work at the time of his death.

Dr. Smith joined the A. V. M. A. in 1935. He is survived by his widow and small child.

B. T. S.

ELMER FRANKLIN HAVEN

Dr. Elmer F. Haven, of Jacksonville, Fla., died unexpectedly on June 18, 1937, of cardiac thrombosis which followed a major operation for prostatic relief. He was doing nicely for a week and expected to leave the hospital within two or three days when the end came.

Born near Marengo, Ill., February 2, 1869, Dr. Haven moved with his parents to the state of Kansas, where he was reared on a farm. He attended common schools and, in 1898, was graduated from the University of Kansas. For some time he was connected with the U. S. Mail Service in Kansas City, during which time he attended the Kansas City Veterinary College. He was graduated in 1909 and then entered the service of the U. S. Bureau of Animal Industry. He was assigned to meat inspection in Chicago, and later was appointed inspector-in-charge at Natchez, Miss. In 1916, he was placed in charge of meat inspection at Jacksonville, Fla., where he remained until he was retired on account of age in December, 1931.

The owner of a nice residential property overlooking a beautiful park and lagoon in Jacksonville, Dr. Haven was happy in his retirement, and his hobbies included flowers, motor travel and the companionship of his wife and many friends. He was an expert carpenter and joiner and thus was able to modernize and beautify his residence.

Dr. Haven joined the A. V. M. A. in 1918. He was a Scottish Rite Mason and his funeral was in charge of the order. Burial was by cremation. He is survived by his widow (née Belle Burnett), three sisters and two brothers.

Dr. Haven was a loyal, conscientious public servant. In his passing the profession lost a valuable asset and the community a fine man and citizen.

E. M. N.

KARL M. OLIVER

Dr. Karl M. Oliver, of Snohomish, Wash., died in a Seattle hospital, July 8, 1937, while under observation for an abdominal ailment of long standing. He was in his 48th year. He was a graduate of the Kansas City Veterinary College, class of 1912, and had been engaged in various pursuits since his graduation. More recently he was connected with the Columbia Packing Company, at Snohomish.

Dr. Oliver joined the A. V. M. A. in 1916 and was active in veterinary association affairs in the Pacific Northwest. He was a member of Alpha Psi fraternity.

JOHN F. COTTER

Dr. J. F. Cotter, of Tabor, Iowa, passed away July 16, 1937, after suffering for several years with a cardiac lesion. He was active professionally until the day of his fatal affliction.

A graduate of the Kansas City Veterinary College, class of 1915, Dr. Cotter built up and maintained an excellent general practice in the territory he served. Endowed with unusual energy and ambition, and possessed of a keen mind and an unusually jovial disposition, he won a large place in his community. As so often happens with live wires in general practice, he filled with distinction a place in the civic and social affairs of his city, as well as serving on the Executive Board of the Iowa Veterinary Medical Association.

Dr. Cotter's popularity in the community he served so well was attested by the extraordinary turnout at his funeral, including almost every practitioner of southwestern Iowa. These colleagues acted as honorary pallbearers. He was buried at Corning, Iowa, and his survivors include his widow, one son, Darrel, a veterinary student at Iowa State College, and one daughter.

A. C. D.

PERSONALS

MARRIAGES

Dr. O. J. Robinson (Mich. '36), of Bad Axe, Mich., to Miss Gurtha Herbert, of Lake Odessa, Mich., in that city, July 17, 1937.

Dr. W. A. McDonald (K. C. V. C. '12), of Baton Rouge, La., to Mrs. Ella Lee Fillastre, July 12, 1937.

Dr. Leonard A. Bowstead (Iowa '37), of DeWitt, Iowa, to Miss Elizabeth Chandler, also of DeWitt, June 14, 1937.

BIRTH

To Dr. and Mrs. I. A. Merchant, of Ames, Iowa, a daughter, Mary Alice, July 13, 1937.

PERSONALS

Dr. Louis J. Payen (O. S. U. '37) is located at Folsom City, Calif.

Dr. Albert Curtiss (Ont. '84), of Holland, Mich., celebrated his 80th birthday on June 14.

Dr. Carl J. Fox (Mich. '26) has purchased an automobile service station at Leslie, Mich.

Dr. Ray D. Hatch (Iowa '37), of Cherokee, Iowa, has entered practice at Prophetstown, Ill.

Dr. T. H. Leenerts (Colo. '31), recently of Pocatello, Idaho, is now located at Amboy, Minn.

Dr. W. M. Vogel (O. S. U. '36) is an assistant city veterinarian of Dayton, Ohio, on a part-time basis.

Dr. J. B. Morse (O. S. U. '35) has opened the Miles Pet Hospital at 13530 Miles Avenue, Cleveland, Ohio.

Dr. R. V. Smith (Ont. '88), of Frederick, Md., is reported to be confined to his home on account of illness.

Dr. Alvin R. Wingerter (Mich. '37) is associated with Dr. W. O. Longfellow (McK. '09), at Fostoria, Ohio.

Dr. Lewis A. Dykstra (Iowa '37), of Monroe, Iowa, has purchased the practice of Dr. F. D. Yeager, at Lena, Ill.

Dr. M. L. Bergsten (K. S. C. '34) has resigned from the U. S. Bureau of Animal Industry and entered practice at Tonkawa, Okla.

Dr. J. H. Muller (U. S. C. V. S. '09), of Sparks, Md., recently suffered the fracture of four ribs while on Bang's disease work.

Dr. B. F. Kennelly (Chi. '17), of Elgin, Ill., has been reappointed McHenry County Veterinarian at a salary of $3,600 per year.

Dr. E. P. Barnes (O. S. U. '32), of Louisville, Ky., has been appointed City Veterinarian, in the Division of Health, of Dayton, Ohio.

Dr. C. L. Sanders (Cin. '16) has resigned as City Veterinarian of Dayton, Ohio, to devote his entire time to his private practice.

Dr. John H. Spurlock (K. S. C. '28), formerly of Trenton, N. J., announced the opening of an office in Allentown, N. J., on July 1.

DR. R. E. LONG (Ont. '12), of Monterey, Ind., had his ankle badly injured when he was thrown by a horse, the latter part of June.

DR. HERMAN J. KROGER (O. S. U. '25) has resigned as City Veterinarian of Dayton, Ohio, to accept a similar position in Saint Louis, Mo.

DR. W. N. ARMSTRONG (Ont. '94), of Concord, Mich., was the principal speaker at the June meeting of the Board of Commerce of Parma, Mich.

DR. R. URBAN TAYLOR (K. S. C. '23), of Oshkosh, Wis., writes that the Oshkosh Veterinary Hospital, destroyed by fire last fall, is being rebuilt.

DR. F. D. YEAGER (Chi. '91), of Lena, Ill., has sold his practice and will take a well-deserved vacation after 46 years of service in his community.

DR. J. R. CORLISS (K. C. V. C. '15), of Newport, Vt., has been reinstated in the U. S. Bureau of Animal Industry. He is stationed at Fryeburg, Me.

DR. ROSWELL L. BROWN (Corn. '35), who has been a member of the staff of the North Shore Animal Hospital, Evanston, Ill., is temporarily located at Pawling, N. Y.

DR. CARL M. OLDHAM (O. S. U. '37), of Charlottesville, Ind., has accepted a position of veterinary inspector with the City Health Department, of Dayton, Ohio.

DR. B. B. HOWELL (Gr. Rap. '10), who has been in state work in Wisconsin for a number of years, has taken over the practice of Dr. J. T. Kennelly, at Juneau, Wis.

DR. H. L. PHILLIPS (U. P. '35), who has been on active duty in the Veterinary Reserve Corps, has taken over the practice of Dr. J. M. Armstrong, at East Providence, R. I.

DR. C. L. BOHAN (Iowa '30), of Bricelyn, Minn., was seriously injured in an automobile accident near Mason City, Iowa, in July. Recent reports are to the effect that he will recover.

DR. THEO. SCHONDAU (U. S. C. V. S. '10) has changed his residence from Thurmont to Halethorpe, Md. He is supervising inspector-in-charge of the Union Stock Yards, Baltimore.

DR. C. L. MCGINNIS (K. S. C. '33), who has been in charge of the Winnetka branch of the North Shore Animal Hospital, of Evanston, Ill., has opened a hospital of his own in Peoria, Ill.

DR. ALBERT M. TAYLOR (Chi. '97) was retired from active service in the U. S. Bureau of Animal Industry, April 30, 1937. He was on the tuberculosis eradication force at Saint Paul, Minn.

DR. CLARENCE N. BRAMER (Corn. '23), who has been associated with the North Shore Animal Hospital, Evanston, Ill., for a number of years, has taken over the hospital and practice of Dr. N. L. McBryde in the same city.

DR. CHELSEA T. PAULISH (O. S. U. '26), who has been in practice with his father, Dr. Wm. F. Paulish, at Franklin, Ind., has accepted a position in the Bureau of Agricultural Economics, and is now stationed in Fort Wayne, Ind., on poultry inspection.

DR. J. I. GIBSON (Ont. '87), of Cedar Rapids, Iowa, was honored by a large group of friends at a barbecue at Blair's Ferry, north of Cedar Rapids, on May 20. The occasion was the 50th anniversary of the graduation of Dr. Gibson from the Ontario Veterinary College. He was presented with a fountain pen and pencil set as a remembrance of the occasion.